Virginia MEDICAL MONTHLY

OFFICIAL PUBLICATION OF THE MEDICAL SOCIETY OF VIRGINIA

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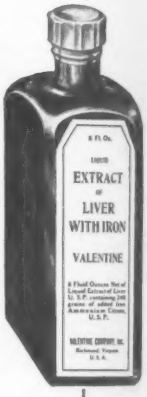

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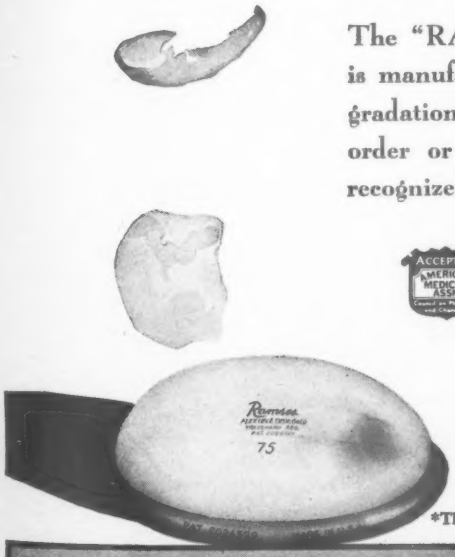

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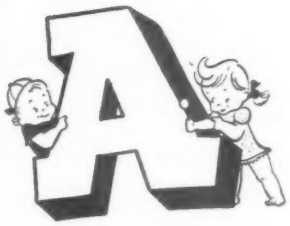

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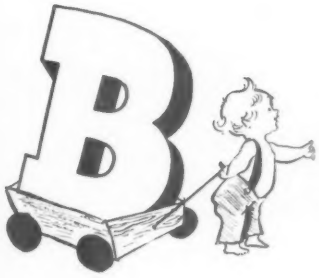

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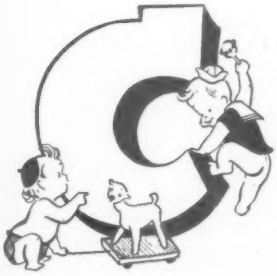

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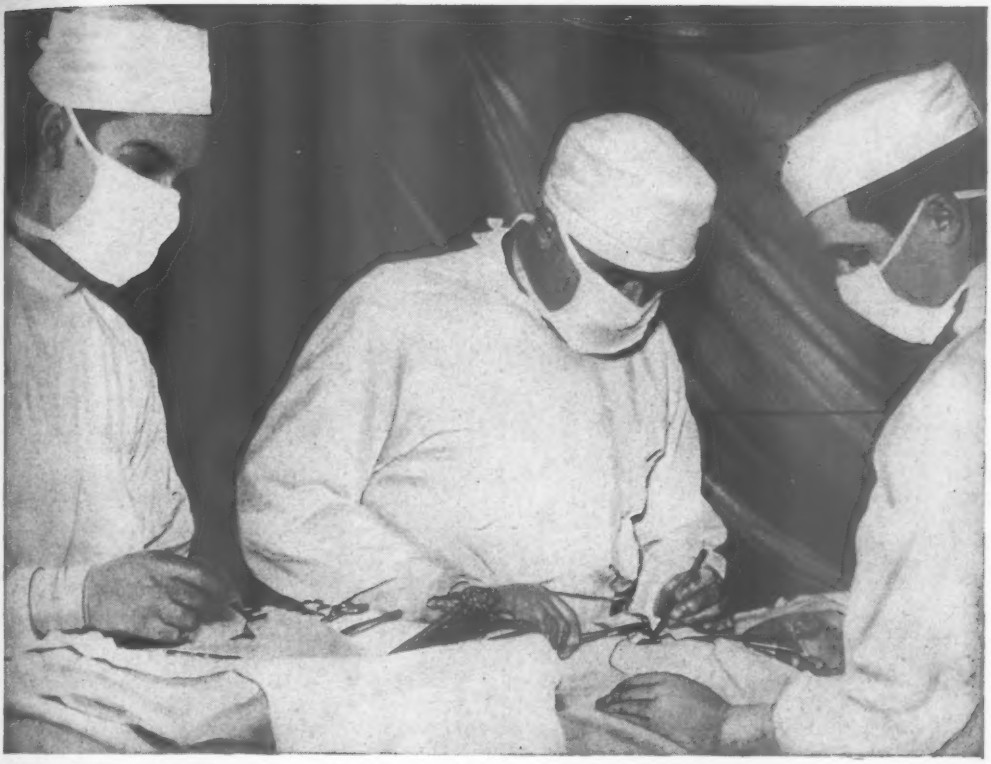

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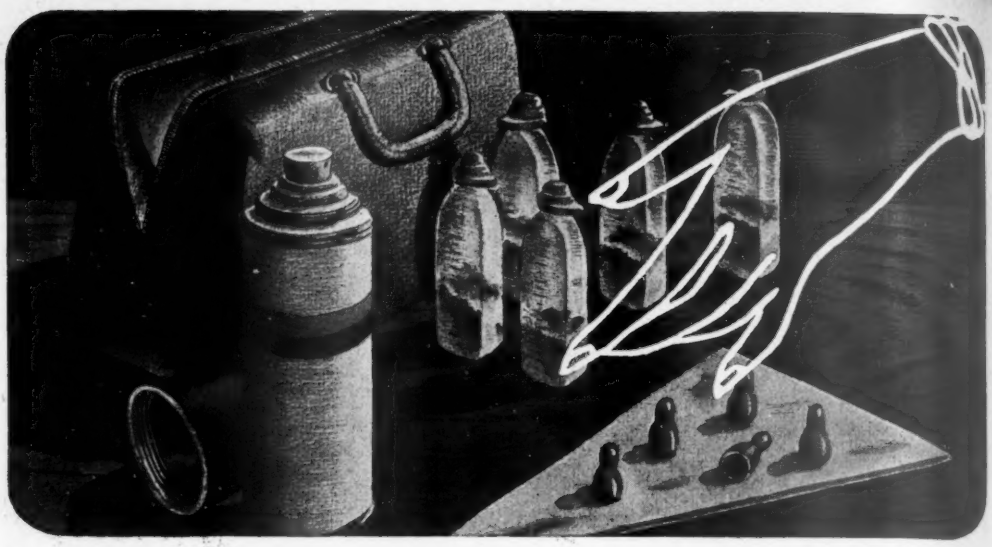

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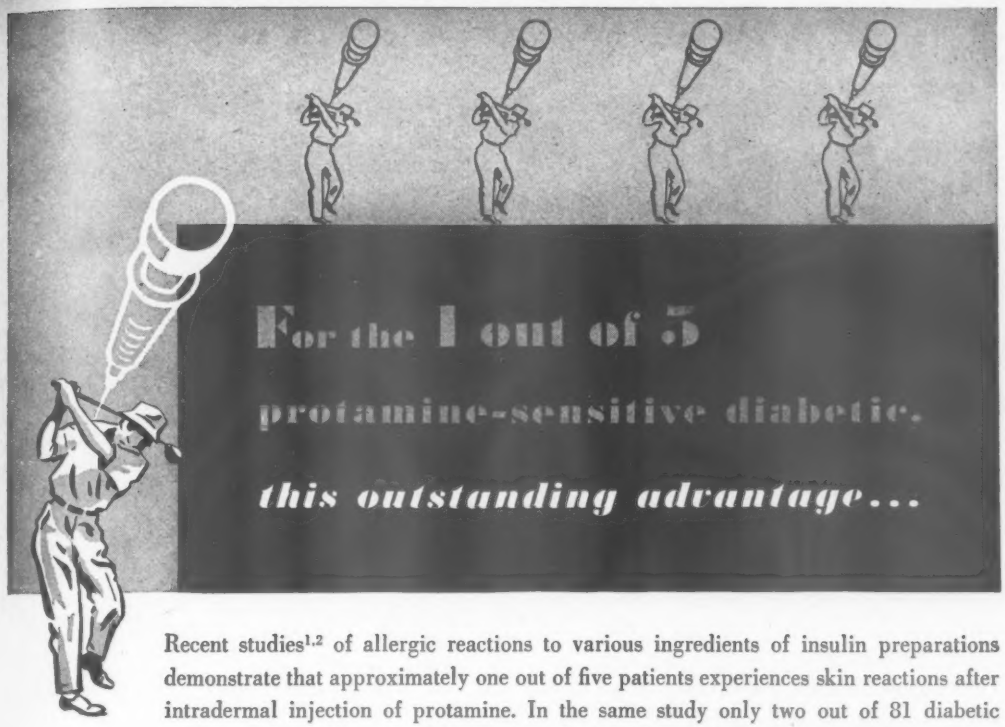

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(1) Page, R. C., and Bauman, L.: J.A.M.A. **124**:704 (March 11) 1944. • (2) Bauman, L.: Bull. N. E. Med. Cen. V:17-21 (Feb.) 1943. • (3) Bauman, L.: Am. J. Med. Sc. **198**:475 (Oct.) 1939, ibid. **200**:299, 1940. • (4) Duncan, G. G., Diseases of Metabolism, Phila., Saunders Co., 1942, p. 782.

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Guest Editorial

Cancer Control and the Doctor

UNTIL specific treatment directed towards the cause or causes of malignant tumors becomes available as the result of fundamental research, any decrease of deaths from cancer must depend largely upon an educational program. Knowledge about the nature of the disease, the clinical course and the available therapeutic resources, admittedly incomplete but growing yearly, is available to the medical profession. The dissemination of a careful selection of these facts among non-medical people has been, for about thirty years, the object of the American Society for the Control of Cancer, now the American Cancer Society, Inc. In Virginia it is now the function of the Virginia Cancer Foundation. The lay population is being taught ever more widely those symptoms that should send a man or woman to a doctor for diagnosis and it is learning in increasing degree the significance of the biopsy and the possibilities of therapy. Although some physicians have criticized this program on the grounds of creating cancer-phobia, those who are closest to it feel that this risk is much less than the risk of ignorance about the disease. Those individuals who will develop unwarranted fear of cancer live in fear of many other of the hazards of living. In spite of some professional opposition the program of lay education has been gratefully received by the population in general. The people of our communities are in fact becoming more and more informed about the disease. Are the doctors meeting the challenge?

That this is not a rhetorical question is obvious to those physicians who deal with malignant disease daily. A proportion of the profession is not alert to cancer or is inadequately informed about it. An instance or so to illustrate this statement may not be out of place. A recent experience concerns a woman who had been taught that a lump in the breast should be investigated. She went to a physician who told her that there was nothing to worry about. He not only failed to meet her intelligent approach seriously but offended her by creating the impression that he felt his patient to be a silly, worried woman. She has since been operated upon for cancer of the breast. Another example is that of a maid in a doctor's home who showed the physician a lump in the breast. He told her not to be concerned about it. A year later she came to a tumor clinic with an advanced inoperable cancer. Within a period of two or three months two patients were seen with cancer of the rectum within easy reach of the examining finger who had actually undergone operations for hemorrhoids not more than a month previously. These may sound like extreme instances. As a matter of fact they are only examples of many such inexcusable blunders.

The situation is not limited to Virginia. In a series of 158 consecutive cancer cases seen in the New Haven Hospital and Tumor Clinic, delay in treatment was due to the physician, at least in part, in 45.2 per cent of cases. Haines and his coworkers (*J.A.M.A.*, Jan. 30, 1943) in reporting these figures state: "The delay by the physician usually was due to poor advice or incorrect diagnosis as a result of failure to carry out the indicated diagnostic procedures." In Nassau County, New York, a lay educational program was initiated in 1928 under the supervision of the County Medical Society. Neff (*New York State Journal of Medicine*, Mar. 1, 1944) has described the

resulting experience. "The first activities of the Committee were the typical ones of preparing educational articles for the local newspapers and sending speakers to address meetings of local organizations of men and women. Almost at once we were met with the embarrassing situation of having patients consult physicians because of symptoms which might mean cancer, only to have the doctors advise them to 'go home and forget it' or 'come back in six months if it doesn't go away.' We thereupon determined that it was of no value to advertise to the public the possibility of cancer cure until more of the medical profession could be gotten into a frame of mind in which they would be willing to meet the patient at least halfway."

It is obvious that a query as to the profession's place in cancer education is not an idle one. Indifference to and ignorance about the disease are deplorable, but not hard to understand. Cancer is an unusual disease in the daily run of cases seen by the family doctor in comparison with the large number of conditions without fatal potentialities. He does not want to be an alarmist; he knows that reassurance is one of his most potent drugs. There need be small wonder, then, that the error of unwarranted reassurance is so often seen. Furthermore, the physician may never have been adequately taught cancer or in the rush of a busy general practice have forgotten most of what he once knew. These are reasons but not excuses. If a physician undertakes the responsibility of the care of the people who trust him, he owes them enough knowledge about cancer and sufficient alertness to its manifestations to realize when reassurance is a breach of faith. We are not discussing errors in diagnosis, to which even the most learned and careful are subject. We are discussing unwarranted ignorance and carelessness, which are equally dangerous. Why should any doctor treat a rectal complaint without a digital and proctoscopic examination? Or abdominal pain without an abdominal examination? Or anemia without an adequate search for a possible malignant cause? These are obviously not common practice, or the medical profession would not stand where it stands today; but they and many similar instances occur.

As the campaign of lay education becomes increasingly effective, the doctor's alertness must become intensified. Even now patients are losing faith in physicians who have failed to recognize the early symptoms and signs of cancer. Lay education must be supplemented by education of a certain portion of the profession; and the informed doctor must play an increasing part in the instruction of his clientele.

This need has been recognized by the Medical Society of Virginia through its Cancer Committee and its Committee for Clinical and Medical Education. The former has established standards for the conduct of tumor clinics and is considering other educational projects. The latter is studying plans to offer after the war a post-graduate course on cancer to all physicians in the state. A portion of the necessary funds is available and the remainder is in the offing. This course will be patterned on the courses previously given in obstetrics and pediatrics. The aim of the educational program for physicians is to diminish the delay between the first visit of a cancer patient to a doctor and the initiation of intelligent and properly designed therapy. The profession of Virginia is facing a growing incidence of cancer. The doctor must not be the one who is responsible for a fatal period of delay in treating the disease. There can be no indifference to the disease that ranks as the second largest cause of death.

EDWIN P. LEHMAN, M.D.,
Charlottesville, Virginia.

EDITOR'S NOTE.—Dr. Lehman is Professor of Surgery at the University of Virginia and Director of the McIntire Tumor Clinic at the University Hospital. He is Vice-President of the American Cancer Society (formerly American Association for Control of Cancer), Director of the Virginia Cancer Foundation, and Chairman of the Cancer Committee of the Medical Society of Virginia.

RICKETTSIAL DISEASES IN VIRGINIA*

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and
KATHARINE ATWOOD WALKE, B.A.,
State Department of Health,
Richmond, Virginia.

Rickettsia is the name given to certain small bacterial forms or bacterial like bodies appearing in the cell cytoplasm and in the intestinal tract of insects and other arthropods and are frequently associated with disease in man. It is not known whether these bacterial like forms are true bacteria or viruses. Many workers have classified them as being in a stage between. They do not pass through ordinary bacterial filters and as yet have been found to grow only within the cytoplasm of the living cell.

Little was known about rickettsiae until the beginning of the present century. Typhus fever, for example, has long been recognized as a clinical entity, but the etiology of the disease remained a complete mystery. Post-mortem findings showed only changes associated with any acute infection. Bacteriologically the tissues were sterile. Finally the causative agent was classified as a filterable

RICKETTSIAL DISEASES OF MAN

GROUP	SYNONYMS	ETIOLOGICAL FACTOR	RESERVOIR	VECTOR TO MAN	FEATURES
Epidemic Typhus Fever	Ship, Jail, Hospital Famine Fever	R. prowazeki var. prowazeki	Man	Lice (P. humanus)	Disease of economic depravity
	Recrudescent Typhus (Brill's Disease)	R. prowazeki var. prowazeki		Not Known	Found in Eastern U. S. Port Cities
	Trench Fever (World War I)	R. wolhynica Syn. R. quintana R. pediculi		Lice (P. humanus)	Tibial ("shinbone") pain
Endemic (Murine) Typhus Fever	"New-World" (U. S.) Sporadic Typhus	R. prowazeki var. mooseri	Rat	Rat Fleas	Mild Disease
	Endemic Typhus of Moscow				
	Toulon Typhus				
Spotted Fever	Rocky Mountain Spotted Fever	Dermacentroxenus rickettsi	Wild Rodents	Ticks (D. andersoni)	Rash on wrists, ankles and forehead first
	Eastern U. S. Spotted Fever	Dermacentroxenus rickettsi	Dog (?)	Ticks (D. variabilis)	
	Brazilian (São Paulo) Fever	R. braziliensis	Not Known	Ticks (A. cajennenuse)	High (70%) Mortality
	Mediterranean (Fievre boutonneuse) Fever	R. conori	Dog (?)	Ticks (R. sanguineus)	"Boutonneuse" refers to nodule at site of tick bite
	South African Tick-Bite Fever	Not Known	Not Known	Ticks A. hebraeum)	Mild disease—Cape-town to Southern Rhodesia
Japanese River Fever	Tsutsuganmushi Fever	R. tsutsugamushi Syn. R. nipponica R. orientalis	Not Known	Mites (T. akamushi)	Regional lymphadenitis
	Trophical Typhus of Ocenia	Not Known		Mites	Sera agglutinates OXK

Fig. 1.

*Read at the annual meeting of the Medical Society of Virginia in Roanoke, October 25-27, 1943.

virus until the work of Wolback and others in 1922 showed it to be a minute intercellular parasite which, with suitable technique, could be demonstrated in human and animal tissues. The first species was

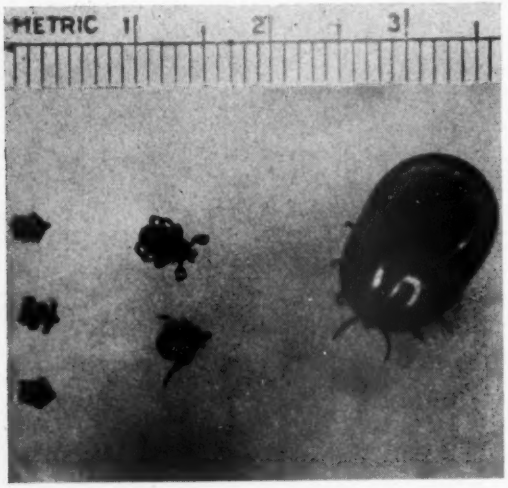

Fig. 2.—Three brown dog ticks, two American dog ticks and an engorged American dog tick.

described in 1916 by da Rocha-Lima who named the bodies Rickettsia prowazeki in honor of Ricketts and Prowazekia, who died of typhus fever.

Rickettsiae appear like very small cocci, diplococci or short bacilli. They are from .3 to .5 microns long and .3 microns in breadth, though some bacillary forms may reach a length of 1.5 to 2.0 microns. Usually non-motile, they are uniformly gram negative and stain poorly with the ordinary stains used for bacteria. The coccidial forms often occur in masses and stain well with Giemsa.

Since much of the work on rickettsiae is still in the experimental stages, it is impossible to make a complete or an accurate classification of the diseases acceptable to all workers in the field. Figure 1 is a greatly simplified classification of the rickettsial diseases found in man to give you a skeleton to which can be added some of the confusing findings now appearing in the literature. It will be seen that there are at least four groups of rickettsial diseases, epidemic typhus fever, endemic or murine typhus fever, spotted fever and Japanese River fever. Since epidemic and endemic typhus fever are the same serologically and immunologically and differ only in vectors and severity of the disease, these two are often put in one group called typhus fever. The second column gives synonyms or names of other diseases which at present appear to belong in these groups. Here you will find many diseases more or less familiar to you. The third column gives the

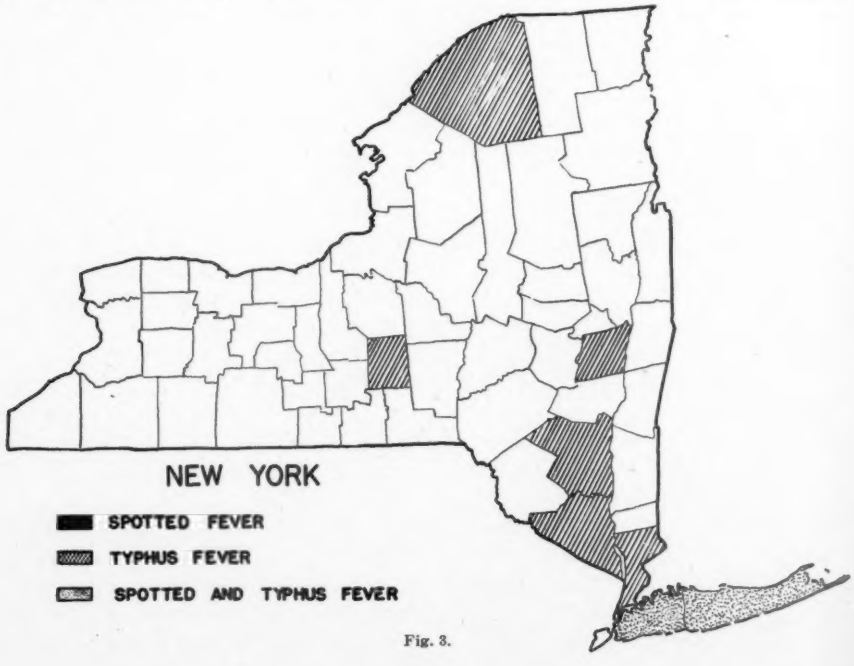

Fig. 3.

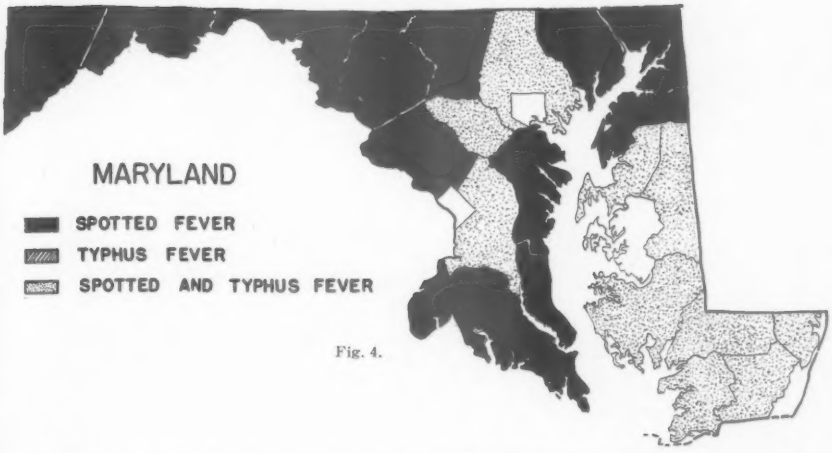

Fig. 4.

name of the organism causing the disease. Some of these have been isolated and have produced the disease in animals and some have not. The fourth column gives the host for the disease, and the fifth the vector to man. It will be noted that roughly each group has a common vector. Epidemic typhus fever is spread by body lice, endemic typhus fever by rat fleas, spotted fever by ticks and Japanese River fever by mites. The last column gives a characteristic of each disease.

To the above list of rickettsial organisms should be added Q fever found in Australia and another described by Dyer in 1938 called Nine Mile fever. Morphologically Nine Mile fever is closely related to Q fever. No cross-immunity exists between these diseases and typhus or spotted fever.

Another disease often associated with rickettsial infections is mosquito borne dengue fever found in the southern or the semi-tropical section of the United States and in tropical countries.

While the subject matter of this paper was being compiled, Doctor Norman Topping published in the United States Public Health Reports a short article on Rickettsioses in India. There it is now known that rickettsiae causing disease entities similar to endemic typhus, epidemic typhus, Malayan scrub typhus or Japanese River fever occur.

Parker states that in many parts of the Rocky Mountain region, febrile reactions, which do not appear to be provoked by a recognized disease, are of relatively frequent occurrence following the bite of the tick Dermacentor andersoni and are referred to as "Colorado tick fever".

Colonel John C. Woodland and others of the Medical Corps of the United States Army have described another entity that they named Bullis fever or Lone Star fever. This is a febrile illness following the bite of the Lone Star tick of Texas and

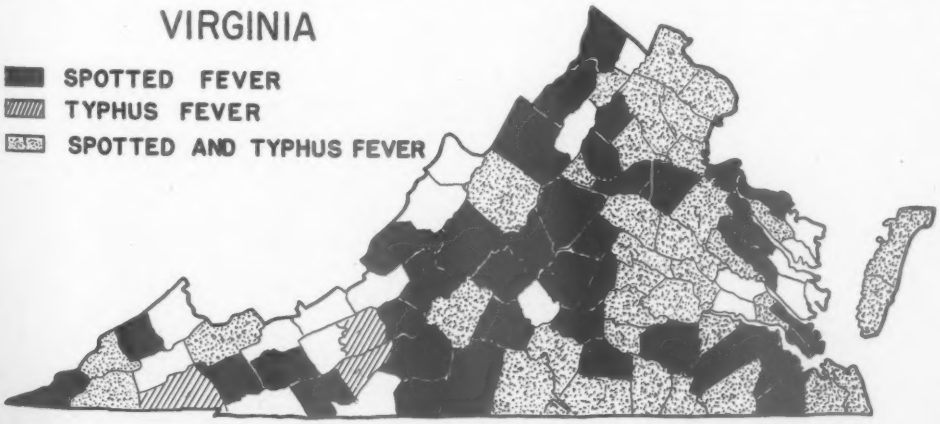

Fig. 5.

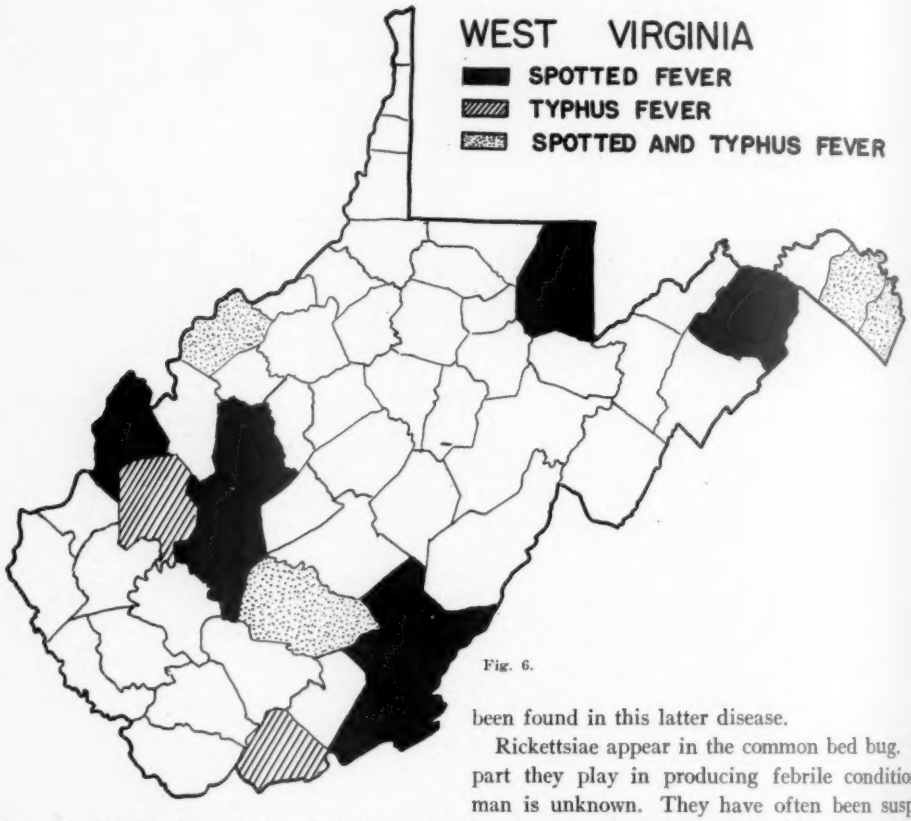

Fig. 6.

resembles Colorado tick fever in that there is a saddle type of temperature curve, no rash, and negative serologic findings. No etiological agent has been found in this latter disease.

Rickettsiae appear in the common bed bug. What part they play in producing febrile conditions in man is unknown. They have often been suspected but never incriminated. A suitable host for culturing these rickettsia has never been found, but it is well known that many persons develop a febrile condition following bites from bed bugs. A rash is not exhibited and the over-all picture is similar to Colorado tick fever as described by Parker in 1937. Shortly prior to his death Dr. W. Brownley Foster independently made a similar observation from his work with cases in Virginia.

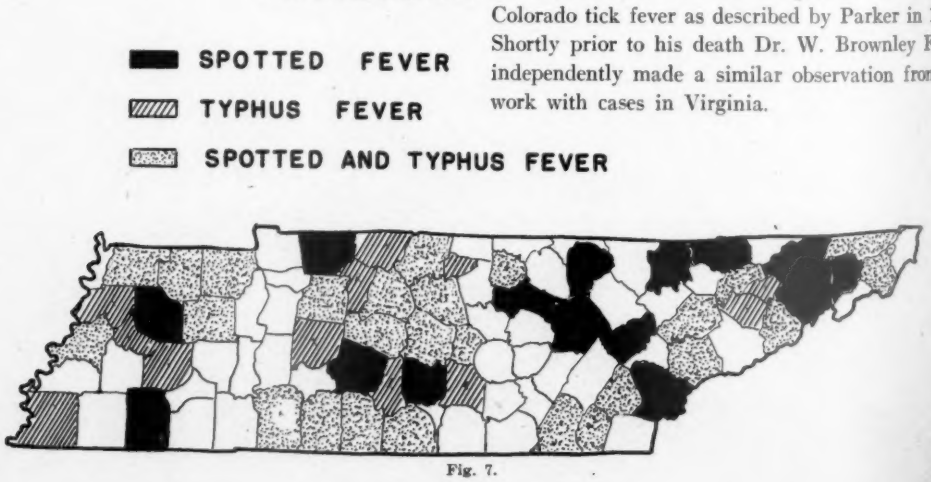

Fig. 7.

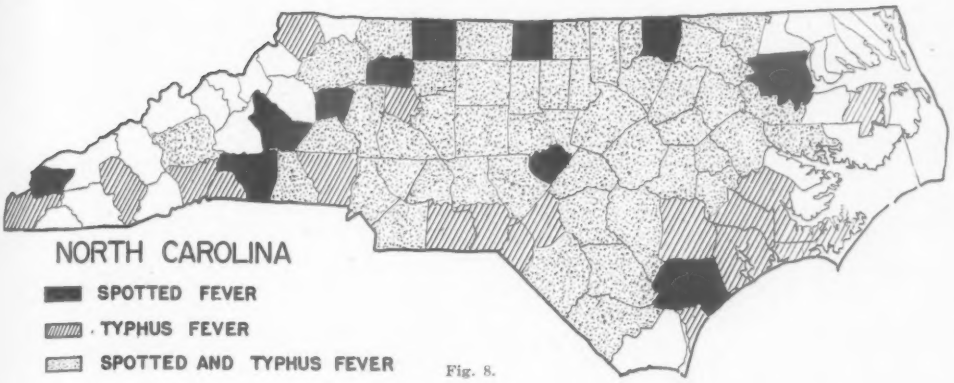

Fig. 8.

Besides these rickettsiae which have been found to be pathogenic, many other species of rickettsia are known which are apparently non-pathogenic. They have been found both in blood sucking and in non-blood sucking insects and inhabit not only the cytoplasm of the living cell but the alimentary tract of the insect itself. Whether rickettsiae are simple commensals of insects or pathogens is not clear.

It can be seen that the human diseases caused by rickettsiae are of world wide distribution. Wherever man, rodents and ectoparasites common to both exist rickettsial diseases will be found. Actually many rickettsial diseases may exist in the same region. They may be recognized or obscure and their existence and characteristics depend on the ectoparasites present in the region. The problem presented by these exotic diseases is fascinating and their ramifications extend far beyond most infectious diseases.

In this paper we will be concerned chiefly with endemic typhus fever and Rocky Mountain spotted fever, the two rickettsial diseases found commonly in Virginia.

At the beginning of this century it was thought that typhus fever had disappeared from the United States and that the cases observed were imported from abroad or from Latin America. In 1910, Dr. Nathan Brill of New York observed a typhus-like infectious disease which he was not willing to call typhus fever. He believed he was dealing with a new disease entity of unknown etiology which was later called Brill's disease. However, in 1912, Anderson and Goldberger successfully inoculated a macacus rhesus monkey with blood from a patient

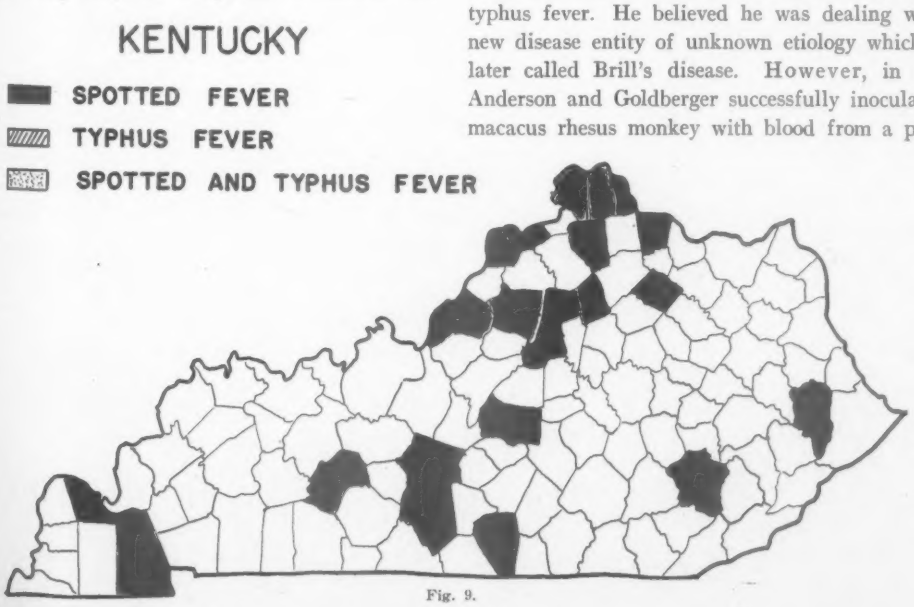

Fig. 9.

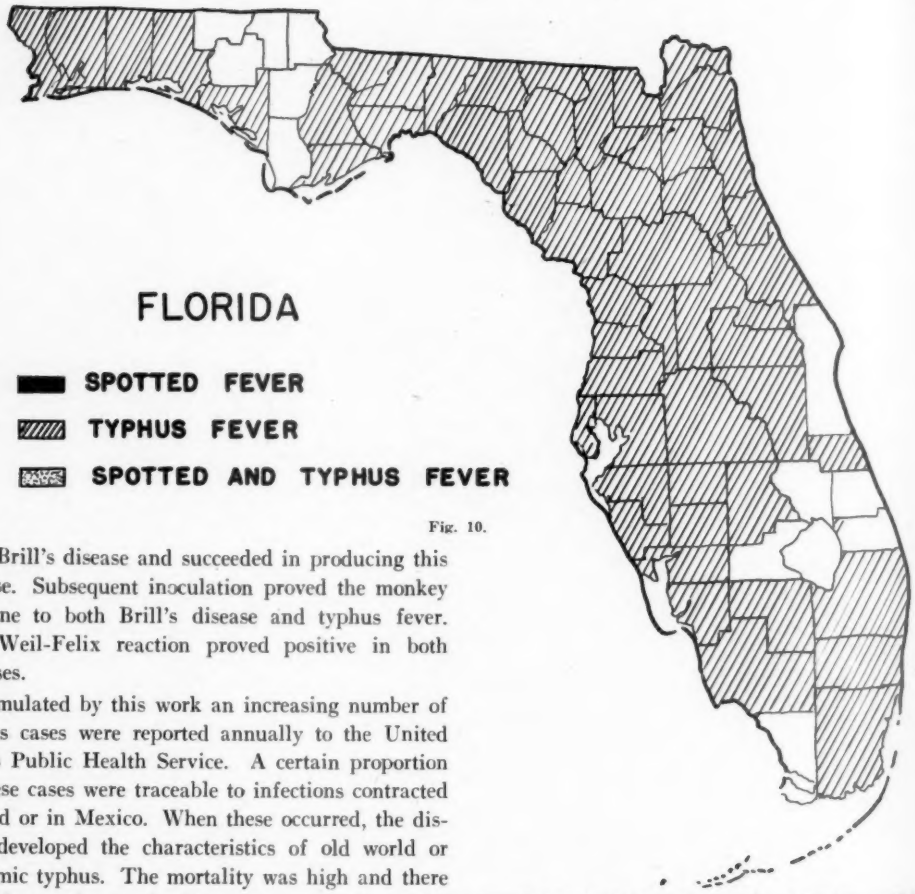

Fig. 10.

with Brill's disease and succeeded in producing this disease. Subsequent inoculation proved the monkey immune to both Brill's disease and typhus fever. The Weil-Felix reaction proved positive in both diseases.

Stimulated by this work an increasing number of typhus cases were reported annually to the United States Public Health Service. A certain proportion of these cases were traceable to infections contracted abroad or in Mexico. When these occurred, the disease developed the characteristics of old world or epidemic typhus. The mortality was high and there was almost always the history of association with louse infested individuals or louse infestation of the patient himself. Most of the cases, however, were the mild type of Brill's disease or endemic typhus, which occur sporadically throughout the Atlantic Seaboard states. In these cases it was rare to obtain a history of louse infestation but they did give a history of contact with rat fleas.

Prior to 1930, Rocky Mountain spotted fever was believed to be found only in the northwest portion of the United States. Its presence there was noted in the literature by E. E. Maxey in 1899. There was one exception, a case which occurred in Indiana in 1925. No other case had been recognized east of a line drawn north and south through the middle of the Dakotas. In 1930, in Maryland and Virginia, Rumerick differentiated spotted fever from endemic typhus fever or Brill's disease clinically and epidemiologically. This observation was confirmed by the laboratory work of Badger. This disease has now been reported in over 30 of the 48 states, in Canada, and in South America, where Sao Paulo Fever is found to be identical with Rocky Mountain spotted fever.

In recent years the rat flea has been found responsible for the spread of endemic typhus or Brill's disease. This disease reaches man from infected rats through the insect vector. Zinsser believed that this infection, an imported and modified form of epidemic typhus, could also be distributed from man to man by the louse.

Three ticks are known to be responsible for the transmission of the virus of Rocky Mountain spotted fever within the United States; the Rocky Mountain wood tick, the American dog tick and the rabbit tick. The first is limited to the West Coast and Canada. The dog tick is widely distributed throughout the Central and Eastern states. The rabbit tick

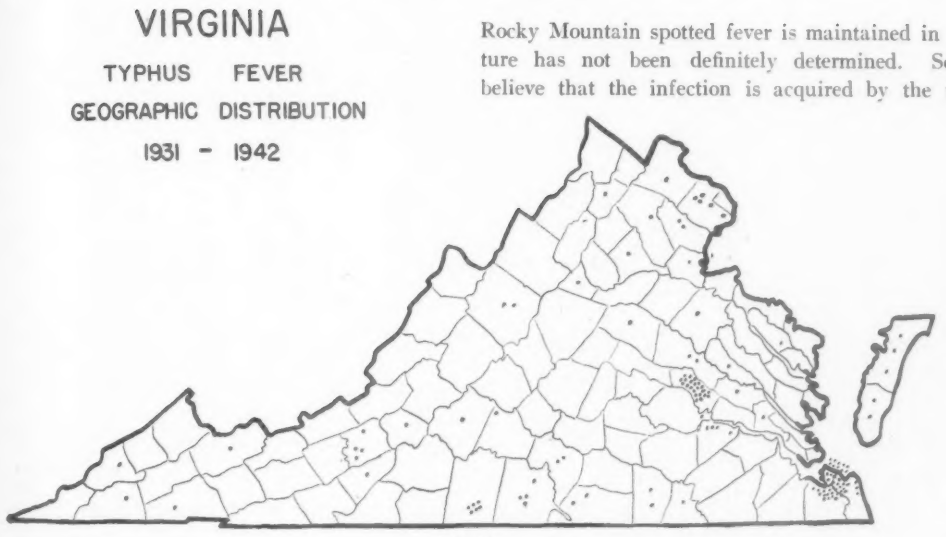

Fig. 11.

is most widely distributed of all, being found from Alaska through the states into South America.

Figure 2 shows the male and female American dog tick which is the insect vector for Rocky Mountain spotted fever in Virginia. Beside them is an engorged dog tick to give you an idea of the relative increase in size after feeding. The smaller ticks are the brown dog ticks found in Virginia in recent years and brought in from Florida. This is the tick which is becoming the household pest.

The actual mechanism by which the virus of Rocky Mountain spotted fever is maintained in nature has not been definitely determined. Some believe that the infection is acquired by the tick larvae or nymphs and passed along to the adult stage. From the infected female it is passed on to the larvae of the next generation or the infected male tick transfers the infection to the female during impregnation. Another theory is that, when the nymphal or adult ticks feed upon susceptible animals, the animal becomes infected. Non-infected ticks, which engorge upon these animals during their period of infection, become infected. They in turn infect other ticks through breeding. It was formerly thought that only larvae become infected

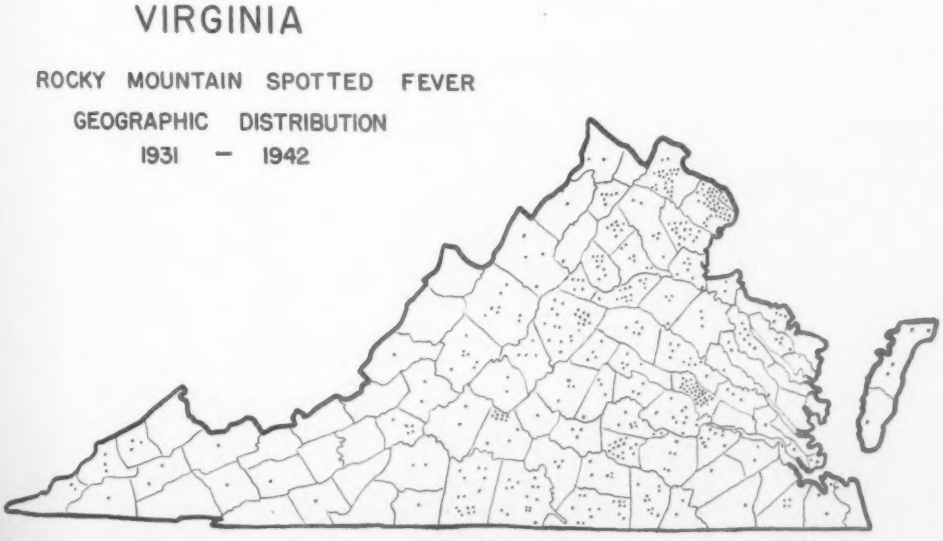

Fig. 12.

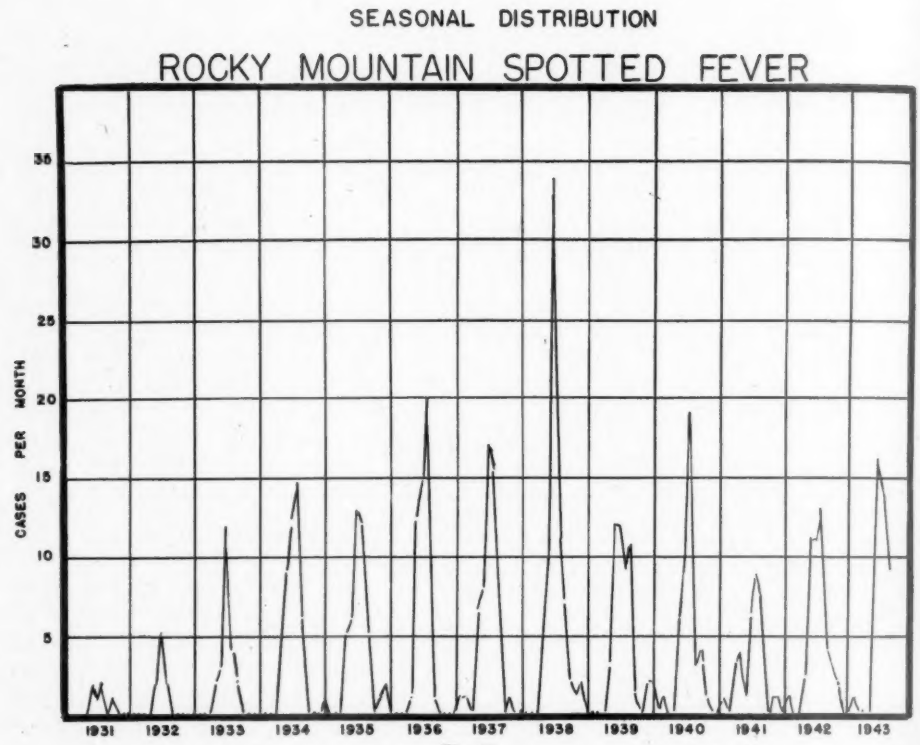
SEASONAL DISTRIBUTION
ROCKY MOUNTAIN SPOTTED FEVER
CASES PER MONTH
35
30
25
20
15
10
5
1931
1932
1933
1934
1935
1936
1937
1938
1939
1940
1941
1942
1943

Fig. 13.

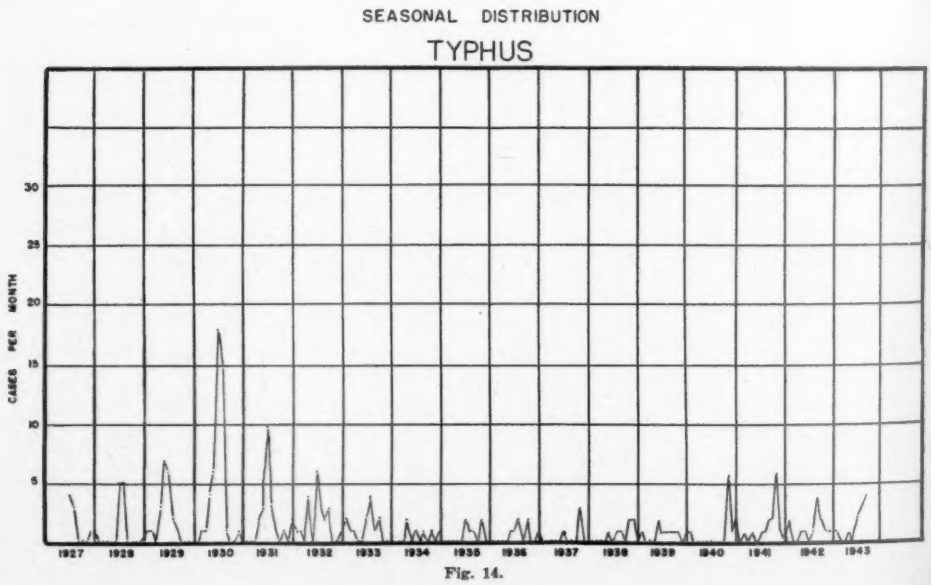
SEASONAL DISTRIBUTION
TYPHUS
CASES PER MONTH
30
25
20
15
10
5
1927
1928
1929
1930
1931
1932
1933
1934
1935
1936
1937
1938
1939
1940
1941
1942
1943

Fig. 14.

DIFFERENTIATION OF ENDEMIC TYPHUS FEVER AND ROCKY MOUNTAIN SPOTTED FEVER

Endemic Typhus	*Rocky Mountain Spotted Fever*
1. Year round.	1. Summer and fall.
2. Urban.	2. Rural.
3. Transmitted by rat fleas.	3. Transmitted by ticks.
4. Incubation period—5-20 days.	4. Incubation period—3-10 days.
5. Mild disease.	5. Severe disease.
6. Temperature lasts 14-16 days.	6. Temperature lasts 21 days.
7. Rash first on body—spreads to extremities—spares face, palms and soles.	7. Rash on extremities first—spreads to body—abundant on ankles, wrists, legs, face, palms, soles, etc.
8. Spleen not enlarged.	8. Spleen often enlarged and tender.
9. Case fatality less than 1%.	9. Case fatality 25%.
10. W. B. C. normal or leukopenia.	10. Leukocytosis.
11. Agglutinates proteus X19 in high titres.	11. Agglutinates proteus X19 in low titres.

Fig. 15.

Fig. 16.

Rocky Mountain Spotted Fever in a woman fifty-one years old who recovered. Photograph taken on eighteenth day of illness. (Photograph, courtesy of Dr. Caroline McGill, Butte, and Dr. R. R. Parker, Hamilton, Montana).

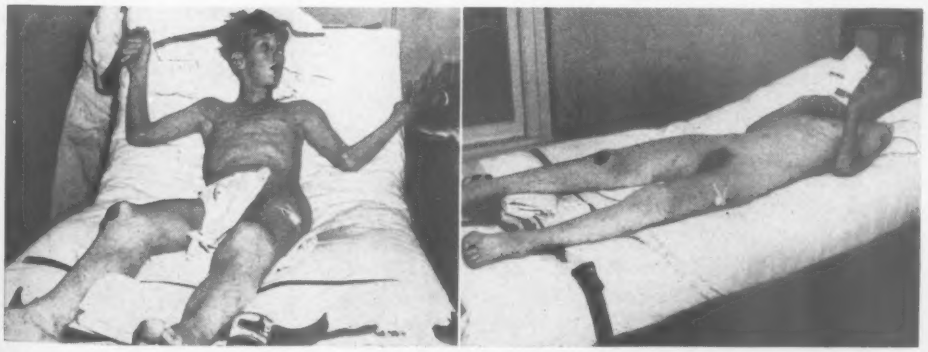

Fig.17.

Rocky Mountain Spotted Fever in a boy aged eighteen years, who expired on the fourteenth day of illness. (Photograph, courtesy of Dr. M. M. Kalez, Spokane, and Dr. R. R. Parker, Hamilton, Montana).

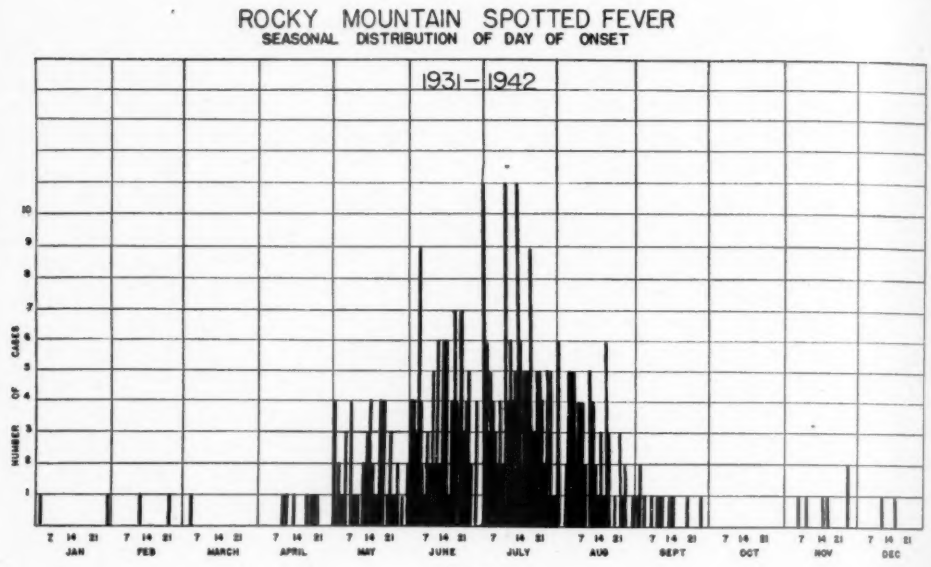

Fig. 18.

in this manner. It is now known that larger animals, such as dogs and sheep, are mildly susceptible and may prove the host for the adult non-infected male and female which usually engorge themselves on larger animals. Little has been mentioned about the role of the common rabbit tick in connection with Rocky Mountain spotted fever. This tick, with the widest geographic distribution, has had the virus of spotted fever demonstrated within it as far north as any known case of the disease in man and far beyond the distribution of the other two ticks. Therefore, it is known that the virus exists in the rabbit tick without the presence of the other two arthropods. The question arises, "can the infection in the other two arthropods exist without the infection in the rabbit tick?"

Many workers have compiled figures as to the incidence of infection in ticks. The work of Parker seems most reliable. He states that the percentage of ticks that carry the infectious virus is frequently less than 1 per cent and rarely exceeds 5 per cent. However, in small localized areas it may reach as high as 11 per cent. Local tick populations, infectious one year, may fail to show infection the following year. The reason for this is unknown. Though as a rule less than one in 100 ticks are infectious, as many as 50 per cent of the eggs from a single infected female may prove to be infectious. The level of virulence remains reasonably constant from year to year. In the fasting tick, the tick that usually is encountered in the Spring, the virus is usually inactive or of the type that causes the atypical cases seen early in the year. Should the same tick be incubated or allowed to engorge itself with blood, the virus becomes reactivated and soon reaches its greatest potential danger.

Before considering the differential diagnosis of Rocky Mountain spotted fever and typhus fever and the characteristics of the disease as reported by the physicians of Virginia, let us look at the distribution of these two diseases along the Atlantic Seaboard. I have prepared a series of eight maps (figures 3-10) which show the counties in eight of the Atlantic Seaboard States which have had one or more cases of Rocky Mountain spotted fever or typhus fever between 1931, the first year the diseases were differentiated, and the present. Cross-hatched counties have had only typhus fever, solid black counties only Rocky Mountain spotted fever and stipled counties both diseases. You will note that the only cases of Rocky Mountain spotted fever reported in New York State were on Long Island. Why this is true, I do not know. In Maryland typhus is found chiefly around Chesapeake Bay, where you would expect it to be associated with the rats on the wharves. In Virginia the same thing is true. A large percentage of the typhus cases are in the shipping centers. In West Virginia, Tennessee, and North Carolina, both

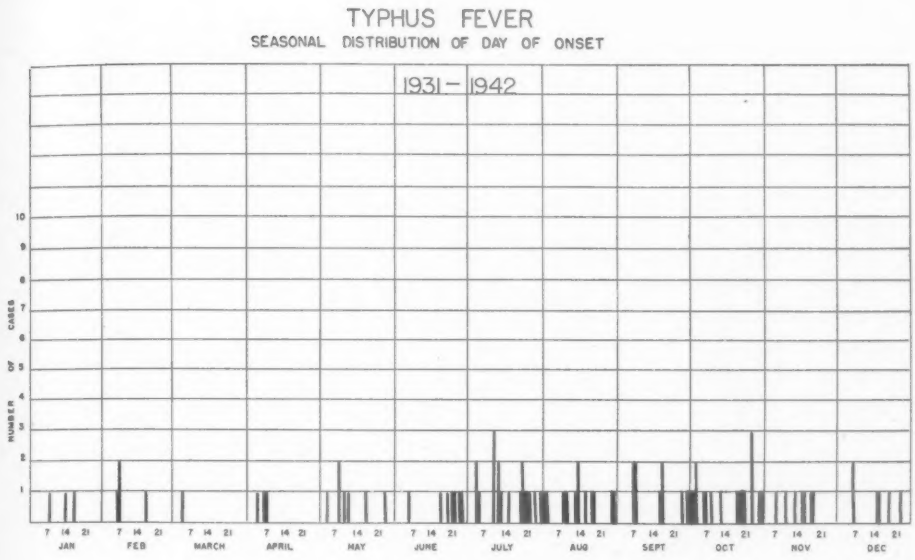

Fig. 19.

diseases are scattered pretty well all over the states in no particular pattern. It is curious to note that Kentucky has no reported cases of typhus fever, although we have just shown that West Virginia on the north, Tennessee on the south and North Carolina and Virginia to the east, do have typhus fever. This leads one to think that possibly typhus fever in Kentucky has been misdiagnosed. In Florida there have been no recognized cases of Rocky Mountain spotted fever, only typhus fever. In general it can be said that Rocky Mountain spotted fever is found in the northern half of the Atlantic Seaboard and typhus fever primarily in the southern half. Virginia and its neighboring states are in the region where both diseases are quite prevalent.

Let us look at another map of Virginia (figure 11), on which the reported cases of typhus fever have been spotted. You will note again that most of the cases were around Norfolk and Richmond. It is because of its association with wharves, ships, large packing houses and the like, that typhus has sometimes been called an urban disease, jail fever or ship fever.

The reported cases of Rocky Mountain spotted fever (figure 12) are scattered pretty well all over the State, although the majority are east of the mountains.

Before going into the differentiation between these diseases, let us look at the distribution of these cases over a period of years. After Rocky Mountain spotted fever was differentiated from typhus fever in 1931, it increased each year till in 1938 there were 72 cases. Since then there have been between 30 and 50 cases reported each year. Figure 13 shows the reported cases plotted by months with the summer months being the peak months. Back in 1927 through 1931 or 1932, when Rocky Mountain spotted fever was not differentiated from typhus fever, typhus appeared to be more prevalent than in recent years (figure 14). Now, of course, we know that probably most of those cases were Rocky Mountain spotted fever. After 1933 there is no peak month for the cases.

The differential diagnosis of rickettsial diseases is often very difficult, because both diseases encountered in the eastern part of the United States are similar in a great many of their characteristics. Rocky Mountain spotted fever and typhus fever are both transmitted to man by the bite of an insect and both of these vectors are found in the mild climate of the Atlantic Coastal States. In neither disease does a lesion develop at the site of the insect bite. Both diseases are acute at their onset and when the disease runs its typical course the duration is often from two to three weeks. There is a similar

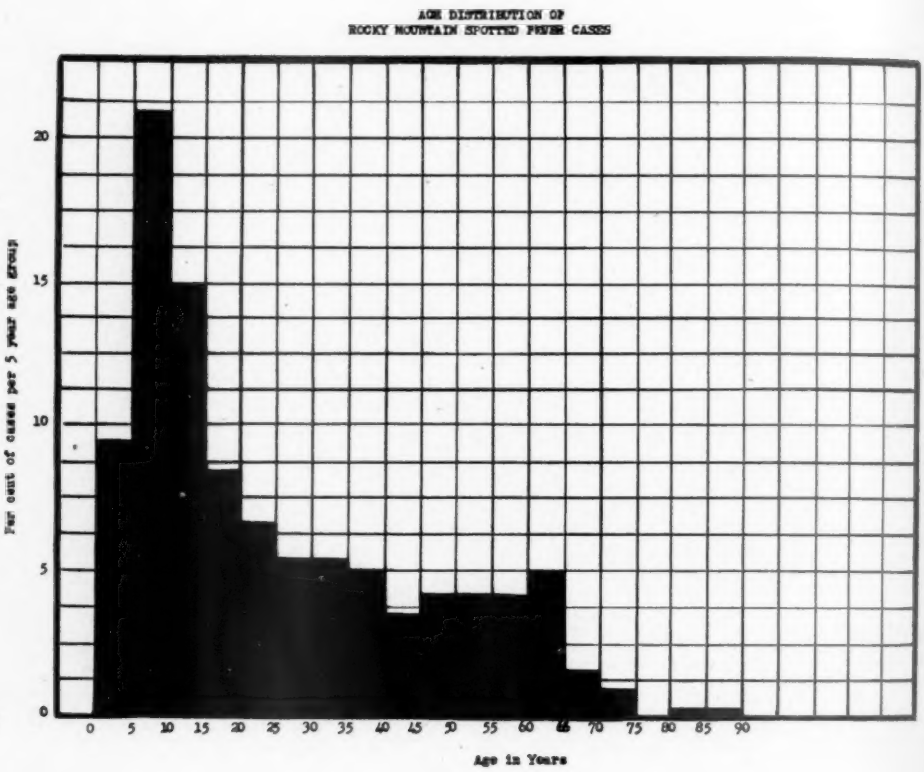
AGE DISTRIBUTION OF
ROCKY MOUNTAIN SPOTTED FEVER CASES
Per cent of cases per 5 year age group
0
5
10
15
20
0 5 10 15 20 25 30 35 40 45 50 55 60 65 70 75 80 85 90
Age in Years

Fig. 20.

history of headache, severe general aching, pains in the muscles and joints, photophobia and fever. Very often when a patient is first examined, the rash is generalized and appears on about the fifth day of the disease. It is red maculo-papular and the lesions are three to four millimeters in diameter and typical flea-bite in appearance. Nervous and mental symptoms are common in both diseases and these symptoms along with the rash indicate the pathology present. Both disease entities agglutinate a proteus organism.

When the two diseases can be separated and studied, variations in their similarity will be noted. Figure 15 shows some of these differences in tabular form. Typhus fever occurs sporadically throughout the year, while spotted fever is limited to the summer months. As one would expect from the vector, typhus fever is primarily an urban disease and spotted fever is predominantly a rural one or has been contracted rurally. Typhus fever is found in all age groups while spotted fever is a disease of young people. Typhus fever is a much milder disease than Rocky Mountain spotted fever. Consequently, the central nervous system symptoms are more marked and permanent damage and sequellae are encountered more frequently in Rocky Mountain spotted fever. The fever lasts longer and the death rate is very much higher. Headache, photophobia and backache have been reported as more severe in Rocky Mountain spotted fever.

The rash in typhus occurs on or about the 7th day of the disease or several days later than the rash seen in spotted fever. The rash in typhus fever appears on the body first and spreads distally, sparing the face, palms and soles while that in spotted fever appears first on the extremities and spreads to the body. With the more severe disease of spotted fever the rash frequently becomes hemorrhagic and necrosis of the scrotum and soft palate occur. A leucocytosis is to be expected. These findings are seldom present in typhus. The spleen is enlarged often in spotted fever but normal in size or not pal-

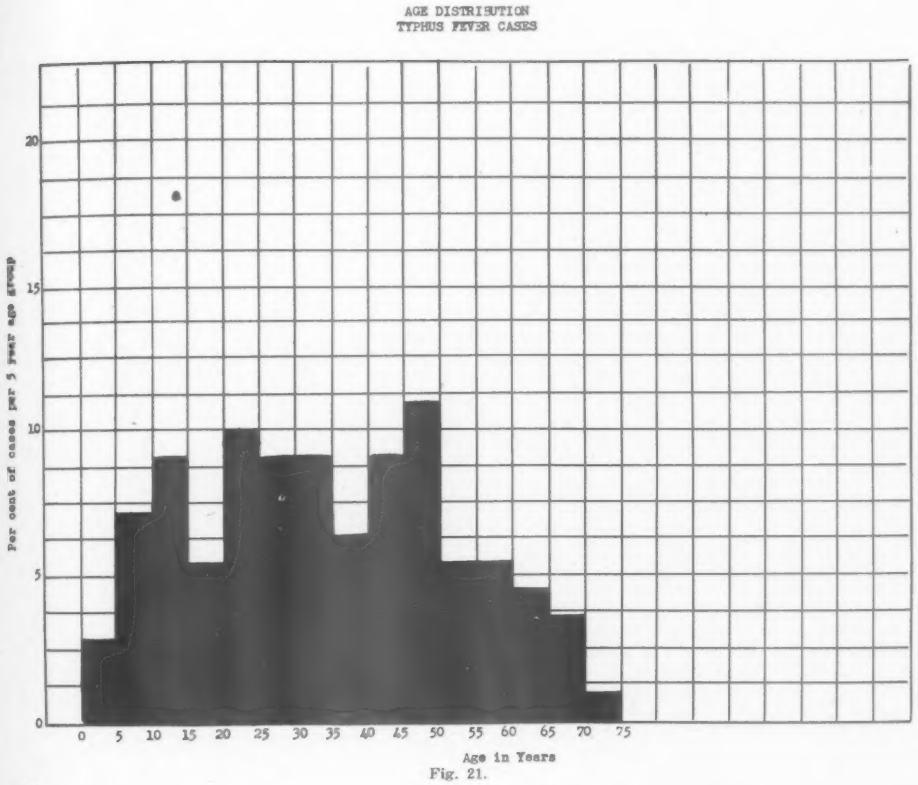

Fig. 21.

pable in typhus. Typhus fever agglutinates the proteus OX19 organism in higher dilutions and earlier than spotted fever does.

Through the courtesy of Dr. R. R. Parker, Director of the Hamilton, Montana, Laboratory, the pictures of clinical cases of Rocky Mountain spotted fever were made available. Figure 16 shows the arms, hands and legs of a woman covered with the typical Rocky Mountain spotted fever rash. Hemorrhagic areas can be noted on the lateral aspect of the left thigh. Figure 17 shows pictures of a boy with Rocky Mountain spotted fever taken a few hours before his death. Note the rash on the palms of his hands, soles of his feet and even his face, and the gangrenous spots on his knees and ankles. We are sorry that we are unable to reproduce in print the excellent colored photographs taken on Virginia patients through the courtesy of their physicians.

The great variation in the clinical picture of these diseases adds to the difficulty in diagnosis. These variations not only are influenced by the individual patient but depend upon the virulence of the virus. Mild atypical cases of rickettsial diseases are frequently encountered in Virginia and they may be so unlike the typical severe cases that they may simulate a different disease.

Malignant and fulminating cases may be confused with typhoid, smallpox, purpura haemorrhagia and severe cases of measles. Proteus cross-agglutination may cause tularemia and undulant fever to be confused with the rickettsiaes. This past year the particularly malignant variety of epidemic meningococcic meningitis has been confused with both Rocky Mountain spotted fever and typhus fever. Many of these cases exhibited a rash, mental symptoms and a degree of illness frequently seen in rickettsial infections.

In addition to these factors there may be biologic variation due to a different arthropod, strain of arthropods, or to a different mammalian host. The differences between endemic murine typhus and epi-

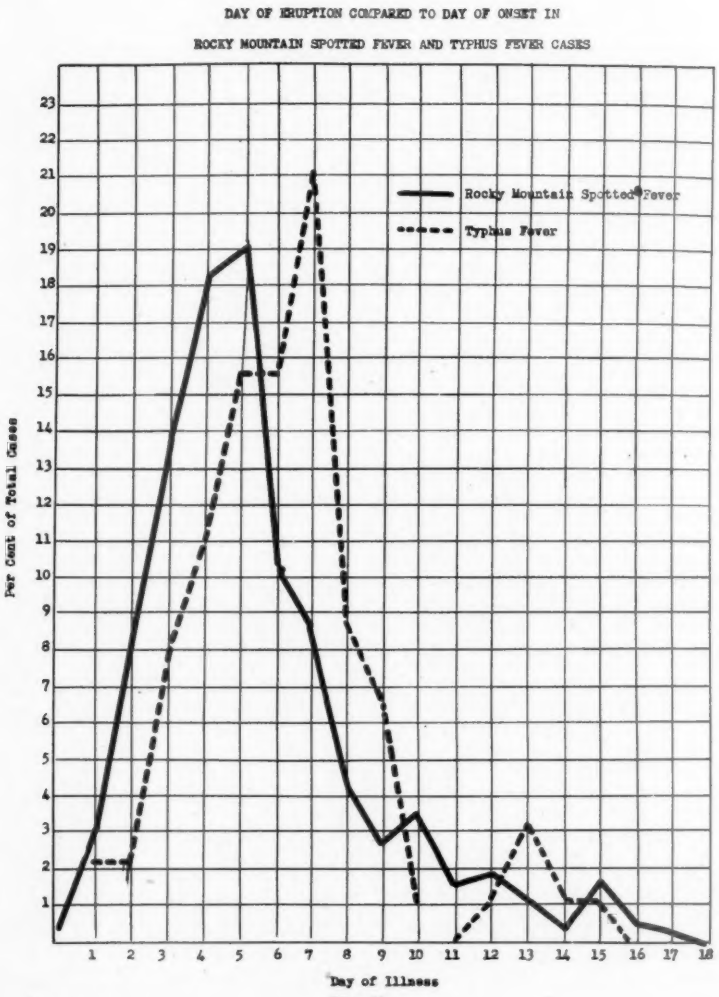
DAY OF ERUPTION COMPARED TO DAY OF ONSET IN
ROCKY MOUNTAIN SPOTTED FEVER AND TYPHUS FEVER CASES
Rocky Mountain Spotted Fever
Typhus Fever
Per Cent of Total Cases
Day of Illness

Fig. 22.

demic human typhus probably may be explained upon this basis.

Since there is so much difficulty in diagnosing typhus and Rocky Mountain spotted fever a laboratory test that was conclusive would be most welcome. None has been found as yet, so the best that can be done is to use the Weil-Felix test in conjunction with clinical findings. The Weil-Felix test is an agglutination test with proteus organisms. Just what the relation is between the rickettsial organisms and the proteus organisms is not known. The proteus organisms do not cause the rickettsial diseases. They have been isolated from rickettsial diseases in many instances, but many more times from numerous other diseases. No one has been able to change from rickettsia to proteus in culture or insect. However, for some unknown reason, the rickettsial organisms agglutinate the proteus. In Virginia agglutination is against O strains of Proteus X19 and Proteus X2. In other localities, the Kingsbury strain is used, but because of cross-agglutination it is not specific. Parker states that an agglutination titre of 1:320 is the lowest that can be considered diagnostic and that 1:160 is highly suggestive. Two or more specimens should be taken and it is to be expected that the first specimen will be negative. Only after the elapse of 10 days or two weeks will the increase in blood titre manifest itself in diagnostic proportions. If,

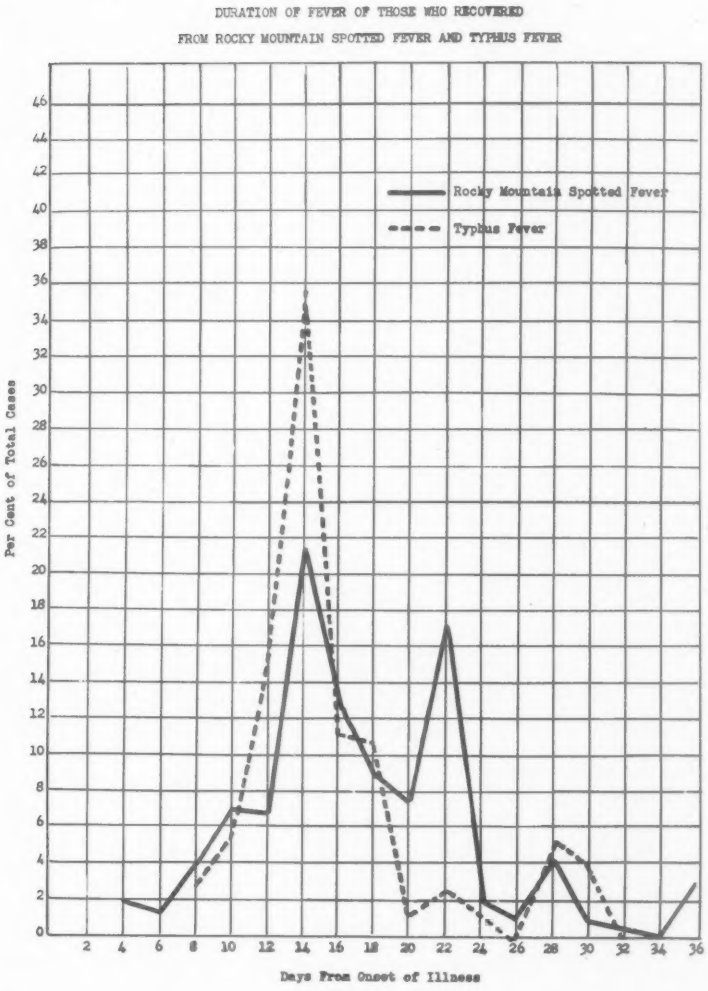
DURATION OF FEVER OF THOSE WHO RECOVERED
FROM ROCKY MOUNTAIN SPOTTED FEVER AND TYPHUS FEVER
Rocky Mountain Spotted Fever
Typhus Fever
Per Cent of Total Cases
Days From Onset of Illness

Fig. 23.

however, the first agglutination was low and the second remains low after two weeks, the possibility of an amanestic reaction brought about by the presence of another disease should be kept in mind. Since tularemia, undulant fever and other diseases also agglutinate the proteus organisms, let me emphasize that this test is meaningless except when used in conjunction with clinical findings.

Since 1927, when Brill's disease or endemic typhus first became reportable in Virginia, the physicians have been asked to fill out a questionnaire on each case they have reported. In this study we have considered only those sent in since 1931, the first year that Rocky Mountain spotted fever was differentiated from typhus fever. From 1931 through 1942, there were 482 reported cases of spotted fever. Questionnaires were received on 435 of these. In the same period there were 133 cases of typhus fever and 109 questionnaires.

The remainder of this paper is an analysis of this data provided by the physicians of Virginia. It took a number of years to get enough cases to be significant, but I hope now they will recognize the importance of their contribution to the study of rickettsial diseases.

Figure 18 shows the distribution of the day of onset of all the Rocky Mountain spotted fever cases. With the exception of a few stray cases in the winter

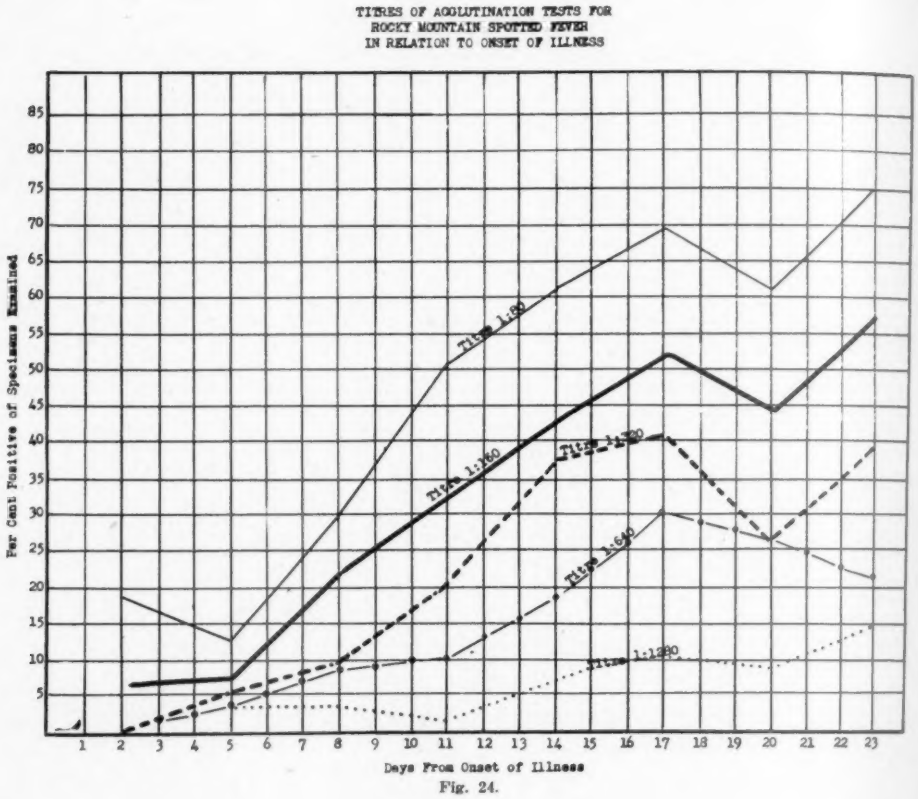

Fig. 24.

months the majority of the cases were in the summer months, May through September, with the peak in June and July. This is in complete agreement with Hull's seasonal distribution of Rocky Mountain spotted fever in eastern United States, shown in his book—"Diseases Transmitted from Animals to Man". The seasonal distribution of the deaths is the same as that of the cases.

Figure 19 shows that the seasonal incidence of typhus fever is not as obvious as that of Rocky Mountain spotted fever. Maxey, in his elaborate study of typhus fever, noted that cases occurred all year but that the maximum incidence was summer and fall, with 70 per cent of all cases falling between June 1 and November 30. The same was found to be true of Virginia cases, with 74 per cent in the months of June through November.

The color, sex and age distribution of the cases reported on questionnaires are of particular interest. Less than 10 per cent of the cases of both diseases were colored, a number too small to be studied separately. In this connection it is interesting to note that Maxey concluded after his study of typhus fever that "the negro for some unknown reason is about exempt". The sex distribution is, however, significant. Fifty-nine per cent of the typhus cases and 63 per cent of the Rocky cases were males. Figure 20 shows the age distribution of Rocky Mountain spotted fever cases. As you can see from the graph, the majority of the cases are in the younger age groups, 21 per cent being in the single age group 5 to 9 years. Hull states that neither sex nor age have any influence on susceptibility to the disease. He says that the differences in age and sex are explained by the greater risk of exposure to ticks, by reason of the occupations of adult males and the more frequent excursion of children into tick infested areas in the course of their play. Figure 21 shows that the age distribution of typhus fever is noteworthy, in that the cases are fairly evenly distributed over the entire life span.

Figure 22 shows the day of eruption compared to

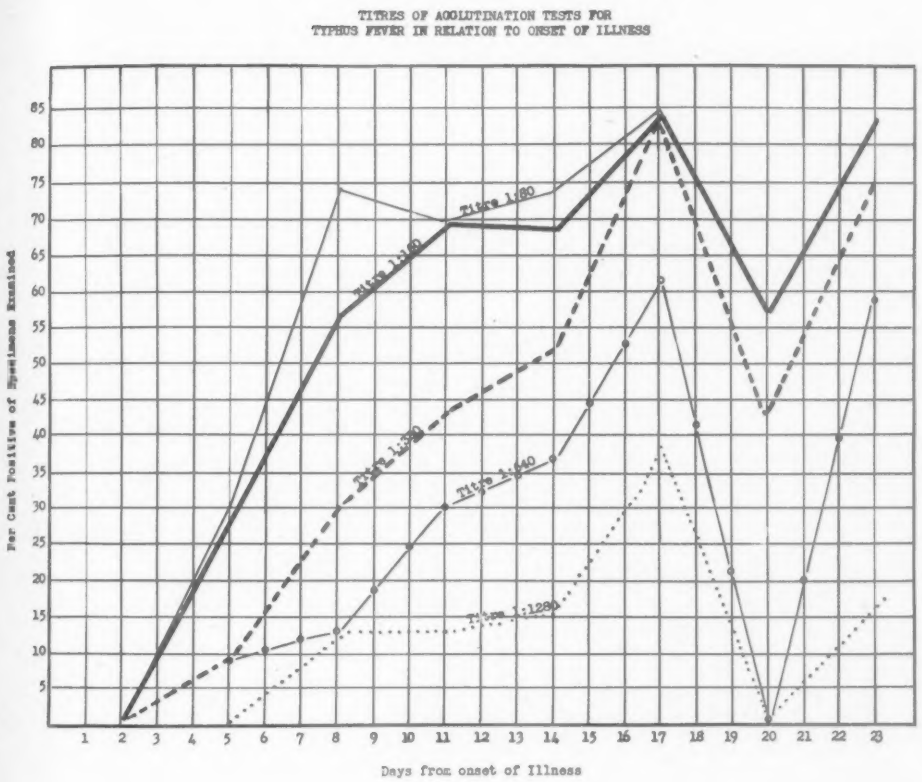

Fig. 25.

the day of onset for both diseases. In Rocky Mountain spotted fever the rash appears earlier than in typhus fever. The average day after onset is 5.5 days for Rocky Mountain spotted fever and 6.5 days for typhus. From the graph you will note that the rash appears on the 5th day in the greatest number of Rocky cases and on the 7th day in typhus cases. There was no difference in the males and females.

Figure 23 shows the duration of the fever of those who lived. The average duration of fever for both diseases is a fraction over 16 days. However, as you can see in the graph more cases of Rocky Mountain spotted fever than typhus fever run a fever for a long time. The average for the typhus cases was brought up by a few cases that may well have been misdiagnosed.

The next three figures are particularly interesting. The data on the Weil-Felix reactions was rather scarce, but, despite the deficiency in data and the probable misdiagnosis of some cases, there is a marked difference in typhus and Rocky Mountain spotted fever. What we did was take the total number of specimens examined in a three day period and see what percentage were positive in five dilutions, 1:80, 1:160, 1:320, 1:640 and 1:1280. Figure 24 shows the results for Rocky Mountain spotted fever. The drop in all the curves on the 20th day is probably not significant but due only to the small number of specimens examined that late in the disease. You will observe that in all dilutions the maximum number of positives is not reached until the 17th day. This is in accord with the experience of others. You will recall, however, that Parker selected 1:320 as a diagnostic titre and 1:160 as suspicious. As late as the 17th day only 41.3 per cent of our cases were positive in the diagnostic dilution and only 52.2 per cent positive in the suspicious dilution. Let us now look at the similar graph for typhus fever (figure 25). Immediately you are struck with the higher percentages positive in every dilution. Moreover, a considerable number are positive in dilutions higher than any

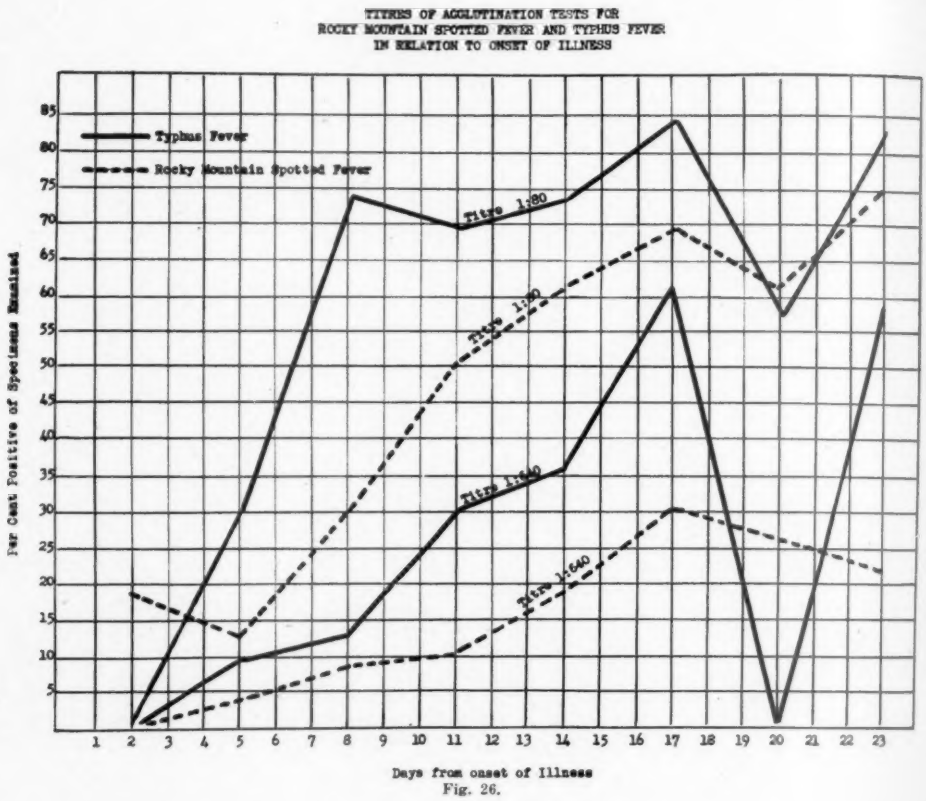

Fig. 26.

shown on this graph. The same meaningless dip as seen in Rocky Mountain spotted fever appears on the 20th day and the maximum again comes on approximately the 17th day. In typhus fever 83.3 per cent of the specimens examined on the 17th day are positive in both the diagnostic and suspicious titres. Figure 26 shows the 1:80 dilutions and the 1:640 dilutions for both diseases on the same graph. The solid line is typhus fever and the broken line is Rocky Mountain spotted fever. This shows how in both dilutions the per cent positive is much higher in typhus fever than in Rocky Mountain spotted fever. For some reason typhus fever agglutinates proteus OX19 in higher titres than does Rocky Mountain spotted fever, but, again, let me remind you that the Weil-Felix test is specific for neither disease and can only be used as an aid in diagnosis.

Among the questionnaire cases, the fatality rate was 15.9 per cent for Rocky Mountain spotted fever and 7.3 per cent for typhus fever. This is a little lower for Rocky and higher for typhus than the figures generally given but not different enough to be significant. For both diseases the average day of death was 13.5, with death falling anywhere between the 5th and the 38th day.

Two hundred and twenty-one, or over 50 per cent, of the cases of Rocky Mountain spotted fever gave a definite history of tick bite. The greatest number were bitten 7 or 8 days before the onset of the disease, with the majority falling between 1 and 15 days. Only 8, or 7.3 per cent, of the typhus cases gave a history of tick bite. Twenty per cent of the cases of Rocky Mountain spotted fever gave a history of rat infestation at home or place of business, while 52.3 per cent of the typhus cases gave a similar history. A negligible number in both diseases had a history of lice or bed bugs.

In conclusion, it can be said that, generally speaking, the data on cases of Rocky Mountain spotted fever and typhus fever in Virginia coincides fairly well with what is known about these two rickettsial diseases.

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DISCUSSION

DR. H. B. MULHOLLAND, Charlottesville: I am sure that all of us will profit by reading Dr. Henderson's paper when it is published, because it is a very excellent survey of the whole question of Rickettsial diseases. We have been particularly interested in spotted fever. I do not think I have seen, or at least I have not recognized, a case of typhus fever recently in the University of Virginia Hospital. Probably we should not see much typhus fever there, because we draw our patients from the country districts, whereas typhus fever patients come mostly from the urban areas.

I should like to ask Dr. Henderson several questions: (1) Whether any survey has been made of the rats in various sections of Virginia to determine the prevalence of rickettsial infestation. (2) Is the reduction in spotted fever cases during the last three or four years due to the fact that a vaccine is being used prophylactically in individuals who must spend a good deal of time outdoors. We have given vaccine to a number of children the past few years. (3) Have you had any experience with spotted fever serum, which has been used experimentally for the past four or five years, and whether you think atabrine, which the Germans use, has any value at all.

DR. HENDERSON, closing the discussion: We have never made a survey of rats to determine whether they are carrying the rickettsia. About the only way to do it would be to test a sufficiently large sample for immunity. We should like to do this at a later date.

In connection with the number of cases of Rocky Mountain spotted fever, I believe we have not reduced the number of cases by free distribution of egg-yolk vaccine, as distributed by the United States Public Health Service. We have for several years distributed this. But when we think of the few immunizations that will be produced, I doubt if it is responsible. Possibly the decrease in Rocky Mountain spotted fever may be due to a decrease in the number of ticks, or some factor with which we are not familiar.

The rabbit serum is prepared at present by Lederle Laboratories. About one-third of the ampoule is given intramuscularly after you test for sensitivity, and the other two-thirds is given after an interval of eight hours. Dr. W. A. Browne, of Alexandria, informed me of one case in which the Arthus Phenomenon had developed in a boy following administration of the serum.

AGRANULOCYTIC ANGINA—A DRUG HAZARD*

ERNEST LEE COPLEY, M.D.,
Richmond, Virginia.

Agranulocytic angina was known to only a few physicians two decades ago. Now it is a matter of informed concern to every doctor. It was inevitable that we would learn rapidly about this new disease for: (1) many cases proved fatal; (2) most cases appeared to be caused by drugs we prescribed for our patients; and (3) the disease has shown a special propensity to kill us doctors. The early observation that physicians, pharmacists and nurses were attacked out of all proportion to their percentage of the whole population constituted a strong clue pointing to some recently introduced drug as the causative agent. Presumably the disease has occurred and killed now and then over a long period of time.

At length, some of its features were observed[1] and thought significant. Then, a few cases were reported,[2] though the symptoms were not fully understood. It remained for the German pathologist, Werner Schultz,[3] after studying a group of six cases in 1922 to recognize the disease as an entity and describe its characteristic syndrome.

Stimulated by his report, a number of others[4,5,6,7] became alert to recognize the disease, and undertook to discover the etiology. Schultz thought it peculiar to women. While all six of Schultz's cases proved fatal, some survivals were reported. It was noted that prostration, high fever and other grave symptoms follow the leukopenia, rather than precede it, as Schultz and others believed.

As to etiology, Schultz could do no better than to call the disease constitutional. He isolated several kinds of organisms from lesions, but could establish no causal relation. Other pathologists[8] found virulent organisms in patients having the disease, reported them as the cause, along with denial of the disease as an entity, and suggested sepsis with granulocytopenia, or agranulocytic infection as the proper name for the disease condition.

When determined, the etiology was as remarkable as the disease itself. Several pathologists, notably Kracke,[9] had suspected the cause to be a benzol-ring drug acting on leukopoietic tissues with a special idiosyncrasy. Following this lead, scores of studies were made and suspicion became conviction.

The earlier studies in etiology dealt chiefly with investigations of amidopyrine. Later it was found that kindred drugs produced the disease. Madison and Squier[10] reported, in 1933, a series of fourteen cases, all of which followed the taking of amidopyrine, alone, or in combination with some other drug. In six cases, in which the drug was continued, there was a mortality of one hundred per cent. In eight, in which the drug was discontinued, there were six recoveries. In two, in which the drug had been taken previously, repetition was followed by a definite fall in the granulocytes. In 1933, Butt *et al*[11] proved that amidopyrine in large doses had a toxic effect on the bone-marrow of dogs. Colmes *et al*[12] showed that a few grains of amidopyrine administered by mouth, or a few milligrams given intradermally, to patients who had recovered from previous attacks induced a severe return within a few hours.

Dameshek[13] studied eighteen cases and decided twelve to be due to amidopyrine, two to allonal, two to dinitrophenol, and one each to arsphenamine and pantopon. It seems significant that in one of the two cases reported by Kastlin,[6] in 1927, six arsenical injections had preceded the onset of the disease. Numerous cases have been reported following the administration of causalin.[14-15] The number of reported cases resulting from the administration of the sulfonamides run into several hundred.

In Jackson's[16] series of 109 cases the use of drugs was blamed as follows: Thirty-four to sulfanilamide, eight to sulfapyridine, thirty-nine to amidopyrine, seven to allonal and four to causalin. The rest were due to various preparations, herein listed.

In the face of the accumulated evidence on the causation of the disease, some cases are still believed to be idiopathic. It may well be that there is an offending drug or agent which has not as yet been determined. FitzHugh[17] states that substances other than drugs—food, bacterial products and metabolites—may produce the disorder through mechanisms of acquired sensitivity.

An incomplete list of the drugs which have been reported or suspected to have caused agranulocytic angina includes amidopyrine arsphenamine, the sulfonamides, causalin, allonal, bismarsen, phenacetin, quinine, cibalgine, allurate, amidophen, cinchophen, neocincophen, amytal compound, dinitrophe-

*Read at the annual meeting of the Medical Society of Virginia in Roanoke, October 25-27, 1943.

nol, sedormid, novaldin, neostibosan, plasmochin, bismuth and nirvanol.

The pathology of the disease is primarily in the bone-marrow. All that follows is secondary. Normally, the red bone-marrow makes erythrocytes, granular leukocytes and platelets. In this disease state the bone-marrow's ability to form granulocytes is greatly impaired or even totally destroyed. As Roberts and Kracke[18] say, "The factory that makes granulocytes goes on a strike and shuts down." The strike, however, appears to be a partial one, for while the bone-marrow ceases to make granulocytes, it continues its normal output of erythrocytes and platelets. These pathologists reported a case studied through two attacks. Having studied the first, they made daily blood counts over a period of time before the onset of the second, and found that the granulocytes disappeared from the blood stream two or three days before the appearance of the clinical symptoms.

The onset of the disease is sudden and stormy. The first complaint is usually of sore throat. Prostration, chill and high fever follow promptly. If the patient does not die in the acute stage, ulcerative lesions develop in the throat, mouth and gums, and sometimes on the vulva and anus. In the early stage the condition may be mistaken for tonsillitis, sore throat or influenza. In some instances, there is a prodromal sore throat; if sore throat is followed by chill and high fever, inquiry should be made regarding the drugs the patient may have taken and a blood examination made.

The cervical lymph-nodes are usually involved, due to the secondary infection of the throat. The spleen and liver may or may not be palpable.

The most significant feature of the syndrome is the blood picture. In no condition are the white cells, particularly the polymorphonuclear cells, so few. The white cells may number less than a thousand, and none of these may be polymorphonuclears.

Agranulocytic angina must be differentiated from lymphatic leukemia, and from overwhelming sepsis. In agranulocytic angina,[19] the anemia is not of high degree; the platelets are normal; it is rare in children, is seen in the aged; many myelocytes appear in the blood stream; and with a temperature of 102 to 104 degrees the patient is acutely ill. In lymphatic leukemia, the anemia is grave, the platelets decreased; it is common in children, rare in the aged; and with a temperature of 102 to 104 degrees the patient may not appear ill. In overwhelming sepsis,[20] anemia, slight at first, is rapidly progressive; many immature white cells appear in the blood stream; the platelets are increased. The main differentiating point is the infectious process which precedes the blood changes.

The reported deaths from agranulocytic angina in Virginia in the past eight years number thirty-six, by years as follows: 1935—5, 1936—1, 1937—12, 1938—2, 1939—1, 1940—10, 1941—4, 1942—1.

Three of the eighteen deaths since 1938 were due to one of the sulfonamides, according to replies to letters sent physicians who reported these deaths. For 1941, Sutliff *et al*[21] reported in considerable detail twenty-eight deaths from sulfonamide toxemia in New York City among 74,553 pneumonia deaths, eight of which were due to agranulocytic angina. The difficulty of obtaining reliable data in cases of death from this cause is obvious. Long's[22] survey of the literature discovered reports of 250 cases of granulocytopenia from sulfonamide compounds. He did not indicate the mortality.

Three cases follow in brief outline.

Case 1. A white man, aged 54, previously in good health, was given amidopyrine in the treatment of influenza, December, 1934. While no symptom characteristic of agranulocytic angina was noted immediately, there ensued a definite decline in well-being, generalized pains, progressive weakness and stiff joints, which at length confined him to bed the most of the time. An anal fissure was the occasion for hospitalization, December 7th, 1935, nearly a year after taking the amidopyrine. On the third hospital day, after cauterization of the fissure, the typical syndrome of agranulocytic angina developed —chill, fever, prostration, coma, and disappearance of polymorphonuclears from the blood stream. The temperature, which was normal on admission, and 101 degrees on the second day, rose to 104.2 degrees and then varied from 102 to 105 degrees for a period of seventeen days. The remainder of the 74 days in the hospital, it was subnormal, normal or moderately elevated. Treatment instituted a week after the acute onset consisted first of 20 c.c. pentnucleotide intramuscularly, b.i.d.; then blood transfusions on the 10th and on the 12th. The blood findings illustrate the course of the disease.

The patient was a semi-invalid after leaving the hospital. The red cell counts varied from three-and-a-half million to four million, the whites from two thousand to four thousand—polymorphonuclears five to six per cent, occasionally as high as twenty

Day	R.B.C.	W.B.C.	Hgb.	Pmn. N.	Pmn. E.	Pmn. B.	Lymphs.	Mon.	L. Mon.
Admission	4,580,000	3,500	85	39	_	_	48	11	_
5th	_____	_____	____	__	10	_	_	42	48	_
7th	_____	3,940,000	4,700	80	7	1	_	42	49	_
9th	_____	_____	14,000	__	4	2	_	36	58	_
12th	____	_____	4,600	__	1	_	_	50	49	_
14th	____	_____	5,200	__	1	_	_	49	50	_
15th	____	4,360,000	2,300	98	12	2	_	32	54	_
16th	____	_____	2,600	__	3	_	_	47	50	_
19th	____	_____	3,600	__	14	_	4	48	29	_
21st	____	5,020,000	5,400	97	28	2	_	48	__	22
27th	____	4,740,000	5,300	94	53	3	_	37	__	7
30th	____	_____	5,500	__	53	_	_	37	10	_
35th	____	_____	6,200	__	39	2	_	39	19	_
37th	____	_____	8,400	__	48	3	_	32	17	_
42nd	____	_____	5,300	__	40	3	1	39	16	_
48th	____	3,760,000	5,600	74	45	2	_	42	11	_
53rd	____	_____	8,500	__	36	2	_	54	8	_
61st	____	_____	7,800	__	34	6	_	47	13	_
67th	____	_____	8,500	__	40	3	3	45	9	_
69th	____	3,680,000	7,400	72	39	4	2	40	15	_

Wassermann Negative.

per cent. He was given, as before, numerous injections of pentnucleotides and also liver extracts parenterally at varying intervals. After one of the injections, twenty-eight months after his first hospitalization, he developed an abscess in the right ischio-rectal region. Again, he was admitted for six days and the abscess drained. Two blood counts were made.

Day	R.B.C.	W.B.C.	Hgb.	Pmn. N.	Pmn. E.	Pmn. B.	Lymphs.	Mon.
Admission	4,080,000	6,900	82	40	3	1	31	25
3rd	_____	4,200,000	7,300	86	47	5	_	36	12

The course of the illness was downhill after that episode. He was given more injections of pentnucleotides, developed another abscess in the left buttock, and was again returned to the hospital two months later for another incision and drainage. Blood counts made at that time revealed a definite anemia as well as more pronounced leukopenia.

Day	R.B.C.	W.B.C.	Hgb.	Pmn. N.	Pmn. E.	Pmn. B.	Lymphs.	Mon.
Admission	3,880,000	3,100	80	20	7	1	42	30
4th	_____	_____	6,000	__	14	1	_	61	24

His condition grew steadily worse after this third confinement. He developed numerous lesions in the mouth, throat, nose and sclerae and the anal region, and open sores appeared elsewhere on the body, particularly on the toes and fingers. The anemia and leukopenia became of severe grade, and the number of polymorphonuclears remained low. All treatment except irrigations and wet dressings to lesions were discontinued. He was admitted to the hospital the fourth and last time in a moribund condition, August 7, 1939, three years and eight months after the onset of the disease. Two blood transfusions on alternate days had no effect whatever on the disease, as the blood counts indicate.

Day	R.B.C.	W.B.C.	Hgb.	Pmn. N.	Pmn. E.	Pmn. B.	Lymphs.	Mon.
Admission	2,640,000	2,100	36	51	2	_	39	8
2nd	_____	_____	1,850	__	53	_	_	40	7
3rd	_____	_____	1,900	40	62	4	_	32	2

The patient died the ninth hospital day. No autopsy was performed.

Case 2. A white woman, aged 60, gave a confusing history: She had cut her finger ten days before, and it became infected. Shortly thereafter she felt generally indisposed and began to have fever and night sweats. During this period she handled a freshly killed rabbit. The relationship of this incident to her sickness was not at all clear.

She did not complain of any particular pain, but she appeared critically ill, and had a temperature of 103.5 degrees. The physical finding was an ulcer on the left fore-finger. She was immediately hospitalized. The first impression was staphylococcic septicemia, the second tularemia. Treatment consisted of codein, wet dressings and sulfadiazine. The temperature was elevated and septic in character the first week, 100 degrees to normal the second, irregularly elevated the third.

One gram of sulfadiazine was given every four hours for the first eleven days. With a decrease in temperature by that time the dose was reduced to half a gram daily for five days. With its increase, however, the dose was doubled and continued until the twentieth hospital day, at which time, the laboratory reported a positive agglutination for B. tularense of 1 to 50. All blood cultures were negative. A total of 108 grams of sulfadiazine had been given and a blood examination at that time showed a red

cell count of 3,800,000 with a hgb. of 71 per cent and a white count of 5,100.

Three days later the syndrome of agranulocytic angina developed. Its treatment consisted of liver extracts parenterally and blood transfusions for three successive days. The disease ran a stormy course to death on the fifth day after its onset. An autopsy was performed.

The blood counts were as follows:

Day	R.B.C.	W.B.C.	Hgb.	Pmn. N.	Pmn. E.	Pmn. B.	Lymphs.	Mon.
Admission	4,700,000	16,400	86	58	..	..	31	11
10th	.....	5,900	70	..	..	..	..	..
20th	3,800,000	5,100	70	..	..	..	..	..
24th	4,300,000	1,350	..	0	..	..	96	4
25th	.....	1,600	..	1	..	..	96	3
26th	.....	3,100	..	0	..	..	98	2

Autopsy diagnosis by Dr. L. J. Buis—Johnston-Willis Hospital:

(1) Agranulocytic angina
(2) Tularemia, splenic (ulcero-glandular type)
(3) Pulmonary atelectasis and congestion
(4) Pericardial adhesions
(5) Uterine fibromyomata.

The bone-marrow from the sternum and lumbar vertebrae was of a bright red color. Microscopic examination revealed absence of mature granular leukocytes, many myelocytes and promyelocytes, and the normal number of red blood cells in all stages of development.

Case 3. A white man, aged 24, an Army Lieutenant, gave the history of having been in a Camp Hospital fifteen days of the previous month, for general indisposition. He had not felt well since. Two weeks later, while at home on leave, he developed a sore throat and high fever. He was given no drugs in the hospital and had not taken any the previous year.

He was acutely ill, with small ulcerative lesions in the throat, temperature 104 degrees, the range for a period of ten days being 102 degrees for mornings to 104 and 105 degrees for afternoons. He was not hospitalized. A blood examination established the diagnosis of agranulocytic angina. Treatment consisted of codein and aspirin the first twenty-four hours; subsequently codein as needed, and a saline mouth-wash and throat gargle.

Recovery took place in four weeks. The blood counts follow:

Day	R.B.C.	W.B.C.	Hgb.	Pmn. N.	Pmn. E.	Pmn. B.	Lymphs.	Mon.	Wass.
1st	4,800,000	2,000	98	2	1	1	40	56	Neg.
3rd	.....	3,900	98	5	2	..	45	48	.....
7th	.....	4,000	95	20	2	2	48	28	.....
20th	.....	4,500	90	10	1	1	60	8	.....
On discharge	.....	5,400	86	46	1	1	52	..	.....

If the tendency of these three cases were uniform or in the same direction, some inference might be made as to the value of treatment. In one instance, however, the patient lived nearly four years with treatment; the second died in spite of treatment, the third recovered without it. They are, therefore, in accord with the various results of therapy reported by authorities[23] in this field, and lend some support to the extreme opinions, varying from disbelief[24] in any form of treatment to enthusiasm[25] for the several therapeutic measures—pentnucleotides, blood transfusions, yellow bone-marrow, liver extracts and adenine sulfate.

I believe Dameshek[26] explains these varying results in his statement, "If the bone-marrow leukocytes are not irretrievably damaged, the patient will recover; otherwise the effects of therapy in a given case are very dubious."

The emphasis is now on prevention of the disease, and this is not without its problems. For some time, hematologists have advised against prescribing amidopyrine or any of its numerous congenors. Three leading drug stores, in Richmond, informed me they are filling a decreasing number of prescriptions for these drugs. The arsenicals and quinine are directed against dreaded enemies, and their value unquestionably justifies the risk of an occasional ill result. The possibilities for evil of all incriminated drugs should be considered carefully before prescribing them.

The reported cases of agranulocytic angina from sulfonamides have been considerable. They have caused also numerous toxic reactions—skin rash, fever, hematuria, oliguria, acute hemolytic anemia, leukopenia; and liver and kidney damage. However, while their benefits have more than compensated for their ill effects, in the light of known dangers they should be administered more discriminatingly. It is wise to use less harmful drugs in non-fatal diseases. The prescribing of one of the sulfonamides for any and all febrile conditions is to be discouraged because

of the reason given, and because of the additional risk of sensitizing a large portion of the population to these potent drugs and so depriving many of their curative powers in future illnesses. Tracy Mallory[27] says, "Do not give me sulfonamide for any non-lethal complaint."

It appears also that the minimum rather than the maximum dose should be administered, and that they should not be given over a long period of time. It is obvious that frequent white blood cell and differential counts should be made on patients receiving them, and the physician should be on the lookout for any indication of an idiosyncrasy. It is generally agreed that, where feasible, tests of degree of concentration of the drugs in the blood are well worth while.

SUMMARY

Werner Schultz, in 1922, recognized agranulocytic angina as a disease entity and described its syndrome. Other pathologists added much to our knowledge of the disease. Its etiology remained a baffling problem for nearly a decade. At length Kracke suspected the disorder was due to an idiosyncratic reaction of the bone-marrow to certain drugs, especially those containing the benzol ring, such as, amidopyrine. This suspicion was verified in several studies.

The pathologic physiology is a destruction of the function of the bone-marrow to make white blood cells, resulting in the disappearance of the white blood cells from the blood stream, particularly the polymorphonuclears.

The distinctive symptoms are sore throat, chill, fever, prostration, the most constant being sore throat.

Thirty-six deaths have been reported in Virginia the past eight years, three of which were ascribed to one of the sulfonamides.

Three cases are outlined briefly. In *the first*, the patient had taken amidopyrine eleven months before the acute onset, which was preceded by a prodromal period of ill health. He was treated with the recommended therapeutic measures—pentnucleotides, blood transfusions and liver extracts parenterally, and lived three years and eight months. In *the second* the disease developed after the taking of sulfadiazine, and was treated with liver extracts parenterally, and blood transfusions. Death ensued the fifth day after onset. In *the third case* no drug was taken before onset which is under the slightest suspicion, nor was any drug given in the course of the illness, yet recovery took place.

These three cases appear to be in accord with the various results of therapy reported by the authorities in this field, and lend some support to the extreme opinions varying from no faith in any form of treatment, to enthusiasm for the different recommended therapeutic measures.

Some means of prevention are suggested. There are indications that physicians have already heeded the warning of hematologists as to the danger of amidopyrine under its various names.

It is suggested that the sulfonamides be prescribed more discriminatingly than heretofore, and in only grave diseases. With assiduous care it is believed many of the ill effects of these valuable drugs can be prevented.

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DISCUSSION

DR. WILLIAM B. PORTER, Richmond: We must appreciate that Dr. Copley has brought to us a very timely discussion. I shall, however, take exception to some of the premises adopted, because I feel that the entity of so-called "agranulocytic angina" has been considerably confused, by including in this syndrome many diseases which should not justifiably be placed there. The question of the causes of leukopenia is still a very difficult one to answer. I think that the essayist has undoubtedly proven beyond a doubt that one of the frequent causes of leukopenia is the use of certain well known drugs. It is not necessary to enumerate these.

On the other hand, we must recognize that leukopenia of an infectious origin, leukopenia associated with the blood dyscrasias, Hodgkin's disease, and miliary tuberculosis are among those very difficult diseases to diagnose and yet are among the most frequent causes for leupopenia. What I wish to emphasize is that the diagnosis of so-called Schultze's disease is a very difficult one; and we should be very cautious, I think, in throwing into this category two of the patients whom Dr. Copley has presented. One of the authors to whom he referred, Jackson, has given sound caution in this connection. He emphasizes that it is very unwise to make the diagnosis of agranulocytic angina if the leukocyte count does not fall below 2,000. In the first case, discussed by Dr. Copley. the leukocyte count was about 3,000. I should be interested in watching the course of this for splenic anemia is a likely diagnosis.

In the second case I should prefer to use the term "sulfonamide idiosyncrasy" rather than agranulocytic angina. In the third case I should much prefer using the term "infectious leukopenia" rather than agranulocytic angina. The case recovered promptly under Dr. Copley's skillful treatment.

Finally, I should like to re-emphasize what Dr. Copley said in the last paragraph of his paper, namely, that the indiscriminate use of these very powerful drugs in patients who have relatively simple diseases is not justified. They should be reserved for patients who have serious maladies.

I am going to differ with Dr. Copley in his statement about the dose of the sulfonamides. It has been emphasized by many authors that the indications for the sulfonamides are pretty clear. Once the decision is made that the patient does have a disease which requires the administration of the sulfonamides, there is a specific dosage which is necessary to control the disease. There is not any excuse for the use of a small amount of the sulfonamide drug because the patient has a mild type of infection and the use of a large dosage when the patient has a severe type of infection. For instance, a pneumococcus infection requires a certain concentration of the drug for the control of the disease. The only difference may be that the patient with the severe type of disease requires intravenous dosage so that the concentration may be built up quickly, whereas the patient with a less severe type may have the slower oral treatment.

I am very grateful to Dr. Copley for bringing us this paper and think he has made a timely contribution to the program.

DR. PAUL D. CAMP, Richmond: I should like to say just a few words and ask Dr. Copley and Dr. Porter whether they have ever seen a case that showed the complications that this one had. This patient was a male between forty-five and fifty who had been perfectly normal. I had made periodic examinations of him. His cardiovascular system was normal. He was definitely an allergic individual. He had a back strain and developed distinct sciatic nerve symptoms. I had a neurologist see him, who prescribed one of the propietary drugs containing amidopyrine. The patient happened to be a pharmacist and, instead of taking the prescribed amount of the drug he took two or three hundred of the tablets over the period of a month or so. He developed, I guess, what you might call agranulocytic angina. Whether he had an idiosyncrasy to that drug or not, he developed the typical syndrome. He was very ill, but, fortunately, recovered. During his convalescence he developed auricular flutter, which was controlled by digitalis. When we were able to get him to the hospital we found he had definite hyperthyroidism, which persisted for quite a while. We treated his hyperthyroidism conservatively. He is now in normal health, except that he continues to have his allergic symptoms, consisting of bronchial asthma, from time to time, and so forth; and he is still not as strong as he was, but his blood picture has returned to normal. It has been checked from time to time and remains normal.

I should like to ask Dr. Copley and Dr. Porter if they have seen auricular flutter and hyperthyroidism developing in the course of this disease.

DR. COPLEY: I should like to ask Dr. Porter to give his experience and his opinion as to the complications of which Dr. Camp spoke.

DR. WILLIAM B. PORTER: I have not seen such a complication. If I had seen it, I think I would ask myself the question if I had not overlooked the hyperthyroidism, instead of its developing afterwards.

DR. CAMP: I might say that there was no clinical indication of hyperthyroidism before. We had never done a basal metabolism on him, because there was no indication for it. He had been checked from time to time thoroughly. I did not previously do a cholesterol nor a basal metabolism because there had been no indication for it.

DR. COPLEY, closing the discussion: I appreciate Dr. Porter's stimulating discussion. It is good practice to challenge diagnoses. We knew from statistics covering

large series of cases that lobar pneumonia carried a mortality rate around 35 per cent before the introduction of the sulfonamides. When physicians in the past, therefore, claimed 10 per cent or 15 per cent, as a result of some particular therapy, I suspected that they had included in their series less fatal respiratory infections. Obviously, studies based upon incorrect diagnoses are of little value.

Manifestly, the diagnosis of blood dyscrasias generally is more difficult than that of some of the more frequently encountered disease conditions. However, leukemia of an infectious origin, Hodgkin's disease and miliary tuberculosis, conditions Dr. Porter mentions. and also lymphatic leukemia are now known to be definite disease entities, and have been thoroughly described by Jackson, Fitz-Hugh, Kracke, Dameshek and others. Furthermore, agranulocytic angina has a more typical syndrome than any of these disorders, and I believe should be differentiated from them without a great deal of difficulty. Certainly, this disease entity need not be confused with either Hodgkin's disease or miliary tuberculosis, because there is no real similarity, either in the blood pictures or the clinical courses.

I am confident the three cases I have described are correctly catalogued. The first was seen by several internists and hematologists, including Dr. Porter's medical staff, and the diagnosis of agranulocytic angina was concurred in. Dr. Porter himself advised an X-ray examination of the chest, apparently to rule out a possible Hodgkin's disease and tuberculosis. Also splenic anemia was distinctly discarded as an explanation of the case. The leukocytes fell below 2000, which Dr. Porter overlooked, 1850 and 1900 respectively on two different counts, and the onset and clinical course were typical of agranulocytic angina.

There is no point in terming the second case, "sulfonamide idiosyncrasy," and this would not alter the pathology involved. Agranulocytic angina is the *result* of an idiosyncrasy on the part of the bone marrow to certain drugs. In this particular instance, the offending drug was sulfadiazine. An idiosyncrasy is a characteristic of this type of bone marrow response, and not a disease in itself. Calling the case "sulfonamide idiosyncrasy" is merely quibbling over terms.

The signs and symptoms and also the blood picture of the third case were thoroughly characteristic of those of agranulocytic angina. Neither was there any infectious process to explain the leukopenia. I am therefore certain all three cases are correctly labeled.

Dr. Porter is correct in asserting that a certain concentration of the sulfonamides is necessary to control pneumonia and other conditions requiring the drug. I believe a concentration from 10 mgs. to 14 mgs. of sulfadiazine per 100 c.c. of blood is required for pneumonia. Whatever dosage, therefore, is necessary to maintain a concentration sufficient to control the pneumonia, obviously, should be administered. What I caution against, is giving the drugs with complete abandon and over long periods of time, when the patient is well out of danger, assuming they were as safe as aspirin. The length of time the drug was given was a factor in nearly every one of the twenty-eight deaths Sutliff *et al.* reported from sulfonamide toxemia in New York. The length of time the drug was administered was a definite factor in the death of the second case I described.

I am very glad Dr. Porter adds emphasis to my contention that physicians ought not to administer the sulfonamides indiscriminately for many of the relatively simple disease conditions which readily respond to less hazardous medication. I have been impressed by the observation that some patients rate a physician's skill on the basis of whether or not he prescribes the sulfonamides. I believe the public needs to be re-educated as to the dangers of these preparations. It is a splendid thing, therefore, that teachers in medical schools are lending their influence in this direction. I do not think amidopyrine should be administered at all.

I want to thank Dr. Camp for his question. The case he describes has the earmarks of agranulocytic angina. However, I have not seen a similar case.

An "Iconography of Vitamin Deficiencies",

Consisting of seven unretouched color photographs revealing symptoms of vitamin deficiency cases that often go unrecognized for long periods have been reproduced and are now being circulated to the medical profession by the Winthrop Chemical Company, Inc. The pictures were originally made at the Nutrition Clinic, Hillman Hospital, Birmingham, Ala., under the supervision of Dr. Tom D. Spies, director.

Accompanying literature points out that many cases of vitamin deficiency may go unrecognized for a long period of time and that diagnosis is particularly difficult in subclinical types since "symptoms are merely suggestive and the physical signs may be minimal". Illustrations, however, it is stated cannot tell the entire story of the consequences of vitamin depletion.

ONE DAY TREATMENT OF SULFONAMIDE-RESISTANT ACUTE GONORRHEA WITH PENICILLIN— A PRELIMINARY REPORT

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Gonorrhea has been a problem for centuries. It is one of the most important causes for loss of time from duty among troops.[1]

The medical profession has been searching for centuries for a perfect therapeutic agent for gonorrhea. Sulfonamides were at first thought to be the answer. Subsequent trial has proved that the sulfonamides are of value but they have their limitations. A drug is desired which will cure the largest number in the shortest period of time.

From recent reports in the literature[2,3,4,5,6,7] it would appear that penicillin fills the need for such a drug. Herewith are presented the results obtained on a series of thirty cases of sulfonamide-resistant acute gonorrhea in the male treated with penicillin at a Military Reservation. Twenty-nine of these cases had had one or more courses of sulfathiazole. One patient was drug-sensitive.

TYPE OF CASES TREATED

1. All patients in this series were adult males and had had acute gonorrhea for periods ranging from 6 days to 3 months (average 25.5 days). All except one had been treated unsuccessfully with one or more courses of sulfonamide.

2. Two had had three courses of sulfathiazole. Eleven had had two courses of sulfathiazole. Seventeen had had one course of sulfathiazole.

3. The average case appeared with an extremely profuse, purulent, urethral discharge. At the completion of the penicillin injections, the urethral discharge had become so scanty that smears and cultures of prostatic fluid had to be obtained. At end of 24 hours there are usually present a clear mucoid discharge obtainable by urethral massage. This mucoid discharge was negative for the gonococcus. By 48-72 hours the mucoid drip disappeared and no urethral discharge or prostatic fluid was available.

All patients were admitted to the hospital for the one day treatment.

THERAPY

1. The penicillin used was supplied in crystalline form in ampules containing 100,000 Oxford units. Before use it was dissolved in 10 cc. sterile distilled water or sterile normal saline. Twenty thousand units (2 cc.) were administered intramuscularly (alternate buttocks) every three hours, for five doses (total of 100,000 U.).

2. Each patient had a smear of urethral discharge taken just prior to his first injection, and this was followed by smears and cultures of urethral discharge or prostatic fluid at the end of 3, 9, 24, 48 and 72 hours. A smear and culture at weekly intervals for next three weeks was performed.

RESULTS

All thirty cases became bacteriologically negative within twelve hours. Fifteen of the thirty cases became bacteriologically negative at the end of three hours; seven were negative at the end of six hours; one was negative after nine hours, and seven were negative at the end of twelve hours.

CRITERIA OF CURE

1. Consistently negative physical and laboratory findings for a period of not less than three weeks prior to discharge from medical observation.

2. A minimum of three consecutive negative gonococcic cultures taken not less than one week apart. The cultural material consisted of urethral discharge or prostatic fluid.

CONCLUSIONS

1. Thirty cases of penicillin treated sulfonamide-fast acute gonorrhea became bacteriologically negative within 12 hours after treatment was begun.

2. Two of the cases had had three courses of sulfathiazole, eleven two courses of sulfathiazole, while seventeen had had one course.

3. One hundred thousand units of penicillin were given in five divided intramuscular doses of 20,000

units each, at 3 hourly intervals, as a complete course of therapy.

4. No toxic effects were noted; blood counts and urinalysis done at 3, 12, 24 and 48 hour intervals were normal; urethral smears and cultures taken at three hour intervals during the course of treatment became negative within a period of twelve hours.

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Winthrop Chemical Company Twenty-Five Years Old.

On June 27, this Company informally marked its twenty-fifth anniversary at a dinner at the DeWitt Clinton Hotel, Albany, N. Y., which was presided over by Dr. Theodore G. Klumpp, president. The company was incorporated in 1919 and derived its name from John Winthrop, Jr., America's first chemist and Colonial Governor of Connecticut. At that time the company offered only 21 drugs, made into a total of 38 different items. Today Winthrop produces more than 150 different drugs, totaling about 750 different items. Penicillin, atabrine, and many other drugs, produced in their Rensselaer Research Laboratories, are serving the armed forces in battle-fronts all over the world. In 1919, the New York office staff numbered only 12; today the executive and office personnel in New York and the branch offices total more than 400. Then, there were about 30 employees in Rensselaer, as compared to 1,550 engaged in the manufacturing plant and laboratories now.

Formation of the Division of Product Development of Winthrop Chemical Company was announced on the 29th. This Division, with headquarters at the Rensselaer plant, will explore the post-war commercial potentialities of products now being supplied by Winthrop to the armed forces, as well as new products developed in their laboratories.

New Books.

The following are recent acquisitions to the Library of the Medical College of Virginia and are available to our readers, the only cost being return postage:

Aldrich, C. A., & Aldrich, M. M.—Feeding our old-fashioned children. 1943.
The American Pharmaceutical Association—The Pharmaceutical Recipe Book. 1943.
American Psychiatric Association—One hundred years of American Psychiatry.
Andral, G.—Medical clinic: diseases of the abdomen. 1843.
Arnett, Alex Mathews—The story of North Carolina. 1942.
Babkin, B. P.—Secretory mechanism of the digestive glands. 1944.
Stern, B. J.—Family, past and present.
Stern, K. and Willheim. R.—The biochemistry of malignant tumors.
Strain, F. B.—Being born.
Swartz, Jacob H.—Elements of medical mycology.
Tenney, H. K.—Let's talk about your baby.
White House Conference on Child Health and Protection—Fetal, newborn and maternal morbidity and mortality.
Wiener, A. S.—Blood groups and blood transfusions.
Wolf, A. W. M.—Parent's manual.
Year Book of Dermatology and Syphilology—1943.
Year Book of General Therapeutics—1943.
Year Book of Neurology, Psychiatry and Endocrinology—1943.
Year Book of Obstetrics and Gynecology—1943.
Year Book of Pediatrics—1943.

TREATMENT OF SULFONAMIDE RESISTANT GONORRHEA—WITH A REPORT OF ELEVEN CASES CURED*

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One of the recent challenges to medicine is the problem of treating the sulfonamide resistant strains of gonorrhea. The sulfonamides have made the treatment of the ordinary case of gonorrhea so easy that both the patient and the physician become very impatient when results are not as expected.

I first encountered this problem in the early days of sulfanilamide. There was no response to two series of 60 grams of sulfanilamide given 2 grams every four hours for three days and gradually reducing that dose. During the brief reign of sulfapyridine I had three cases that did not respond to this drug but which were cured by sulfanilamide.

With the advent of sulfathiazole the resistant cases became more rare and I did not encounter another until after sulfadiazine was on the market. I successfully alternated the sulfathiazole and sulfadiazine until Case A reported. On this case nothing but time was successful.

On the next resistant case the patient asked for a solution of permanganate of potash for urethral instillations and I decided to use a local solution of sulfanilamide, 7.5 gr. to an ounce of boiling water. At this time I also gave this patient .1 cc. of gonococcus filtrate intracutaneously. Improvement was marked but not rapid. After treating three cases by this method with successful results, I asked Dr. C. C. Haskell for a solvent that would allow stronger instillations. He suggested that a suspension might prove more effective than a solution and showed me a reported cure in about four weeks in which the duosulfon alone had been used. My last seven cases were treated with the duosulfon suspension instillations together with gonococcus filtrate given every three days when possible.

I consider the technic of instillations important. The patient is instructed to void, then wash out the urethra with two syringes of hot water. The duosulfon suspension is then well shaken, instilled into the urethra and retained for three minutes. This is done three times daily.

In the case reports herewith the following abbreviations are used: Gonococcus Filtrate—GCF; Sulfathiazole — St; Sulfadiazine — Sd; Sulfanilamide—Sa.

Case A. W. A. S., white, male, 26 years. Exposed six days before reporting. Purulent urethral discharge noted that morning. No pain or burning. Laboratory report positive for Gram-negative intracellular diplococci. Patient was started on St gm. 2, and one gram every four hours. In three days there was no noticeable change. The treatment was changed to Sd with the same dosage for four days with no change. Sd continued for one week with no change. St was given for one week with intracutaneous GCF every three days with no improvement. GCF was continued for five doses and urine was alkalinized after the week of St. There was no improvement. Ascorbic acid was given orally 500 mg. daily for three days and 300 mg. daily for one week; there was no change. The alkaline treatment was resumed and St 2 gm. every four hours was given for two days with no change. Sd was repeated for one week with no change. Acriflavin and potassium permanganate solution instillations were alternated until the patient finally ceased returning for treatment and said that he was well.

Case 1. R. A. C., white, male, 19 years. Had a history of four exposures in past two weeks and reported with a purulent urethral discharge and persistent pain and burning. The laboratory report was positive. Treatment was started with St 2 gm. and 1 gm. every four hours. At the end of four days there was no change. Sd was given for four days in the same dosage with no change. St was given for two days 2 gm. every four hours with no change. This patient had heard of potassium permanganate instillations and requested this drug. I gave him the solution of Sa mentioned above and started GCF injections in the forearm. After the second GCF considerable improvement was noted. He became symptom free on the tenth day of this routine. A total of five injections of GCF were given and on the twentieth day his prostate was examined and found normal. A smear was made of the expressed secretion and was reported negative. I could not

*Read before the Warwick County Medical Society, May 9, 1944.

check this patient later as he was a soldier and was transferred.

Case 2. W. D., white, male, 22 years. This patient was brought in by Case 1 and thought that the source of infection was the same. He had been taking Sd 1 gm. four times daily for one week and had noticed no improvement. Laboratory report was positive. St was given 2 gm. at once and 1 gm. every four hours for four days with no improvement. GCF injections and Sa solution instillations were started. Improvement was noted after the second GCF. Treatment was continued for two weeks with five GCF injections. Discharge stopped on the seventh or eighth day. There was no recurrence after a drinking party during the first week after completing treatment. One week after treatment stopped the prostate was normal and secretion negative for pus or organisms.

Case 3. W. R., white, male, 23 years. Reported with typical symptoms and a history of eight prescriptions for large white tablets of two different kinds within the past three months. He had noticed no improvement in the urethritis, but was feeling so badly that he had missed several days from work. The laboratory reported no intracellular diplococci. GCF and Sa solution were started and the patient stated that the discharge stopped the day after the second GCF injection but reappeared the next day when he indulged in alcoholics. This patient was symptom free again on the eleventh day and remained so until his final check-up on the twenty-second day, at which time he was negative in every respect.

Case 4. J. R. F., white, male, 26 years. This patient was an officer at one of the Army Posts and had access to the sulfonamides at the Post Hospital. He had treated himself with Sd in adequate dosage and time schedule for one week and had then tried St for five days with no noticeable improvement. I started this patient on GCF and duosulfon suspension instillations. He could not report regularly for the GCF injections but received a total of four. He became symptom free after the second injection. He continued the instillations for another week and was negative on check-up eighteen days after receiving his fourth GCF treatment. He returned six weeks later prior to a transfer and was negative at that time.

Case 5. S. C., white, male, 27 years. This patient reported with a rather thin urethral discharge which was reported positive by the laboratory. He had acute gonorrhea five months previously and the discharge stopped after about two weeks of oral St. He had a recurrence of the discharge eight days later and the thin discharge has persisted with occasional two or three day remissions since that time in spite of continued oral sulfonamides. The prostate was markedly enlarged and boggy and the prostatic discharge was thicker than he had been observing. The prostate was gently massaged and the patient given GCF and duosulfon suspension instillations. He received a total of five GCF injections and discharge stopped after third. He continued instillations and massages for one month, at which time prostate was firm and secretion negative. He returned for check-up after four months. Prostate was enlarged moderately and secretion was negative. He reported an occasional morning drop.

Case 6. W. R. O., white, male, 34 years. This patient reported with a history of positive laboratory report for gonorrhea and treatment with only sulfadiazine for two months. My laboratory report was negative. He had a heavy discharge and complained of severe pain at times. He was given St for four days without change. GCF and duosulfon were started and the discharge stopped on the seventh day. Treatment was continued for five GCF injections, at which time he was completely negative.

Case 7. R. S. S., white, male, 20 years. Reported with history of acute gonorrhea of two months' duration. Treated extensively with Sd and St. In spite of treatment had developed orchitis on the left with swelling the size of a large hen's egg. Had been unable to work for past two weeks, so came home for treatment. GCF and duosulfon started and GCF repeated in three days. Patient disappeared for thirty-two days. When he returned there was no discharge and the left testicle was down to almost normal size. There was no pain on pressure along cord or epididymis. His prostate was normal and secretion negative. He had continued instillations.

Case 8. M. H., white, male, 26 years. Patient referred as sulfonamide fast by V. D. Clinic. Purulent discharge of four weeks' duration. Had positive laboratory report at Clinic. Had received St and Sd each for one week without improvement. GCF and duosulfon started. Discharge stopped after second GCF injection. Unfortunately the local supply of GCF was exhausted. Discharge returned

three days later. It was three weeks before more GCF could be obtained. During that time old typhoid vaccine was given, hoping that a reaction to foreign protein would suffice. Nothing was accomplished constructively and his disposition was much worse. When the GCF was available the discharge stopped on the fifth day and the patient received four injections in the second series. He was negative on discharge.

Case 9. J. B., white, male, 33 years. Patient referred by V. D. Clinic with history of onset with positive laboratory report two weeks before. Had received St and Sd each for one week with no improvement. Laboratory report was positive the day he reported to me. GCF and duosulfon started. Discharge stopped two days after first treatment. A total of four injections were given in fourteen days, at which time he was negative. This is the most rapid recovery I have noted.

Case 10. M. G., white, male, 20 years. Patient had acute onset three months before after a three day incubation period. He took nearly two hundred sulfonamide tablets with no change. He reported to the V. D. Clinic where he was found positive and received one week's treatment with Sd with no change. GCF and duosulfon were started and the discharge stopped on the third day. A total of four GCF injections were given and the patient was negative.

Case 11. W. A. S., white, male, 19 years. Patient reported with purulent discharge, positive report, and a box of Sd which he had been taking six days. St was given on usual schedule with no results in three days. GCF and duosulfon were started. Discharge stopped on fifth day. Patient disappeared after fourth injection, leaving an unpaid bill and an incomplete record.

DISCUSSION.

In my experience, if any sulfonamide given orally for gonorrhea does not produce noticeable improvement in forty-eight hours, it will not cure that given case. Frequently a change of drug will be effective. Neither GCF or duosulfon suspension instillations alone have been effective, but by synergistic action are very successful.

Masonic Temple.

Tantalum to Be Available to Civilian Surgeons.

Tantalum plates, foil, screws and wire to repair broken bones, nerves and skulls will shortly be available to civilian surgeons through a recent allocation of the War Production Board, according to an announcement made by Dr. Gustav S. Mathey, President of the Johnson & Johnson Research Foundation, New Brunswick, New Jersey.

The Johnson & Johnson Research Foundation is a non-profit organization, founded in 1940 to endow research in universities and hospitals and to disseminate summaries of findings to members of the medical profession. Dr. Mathey states that by an agreement between the Ethicon Suture Laboratories, Johnson & Johnson subsidiary, and the Fansteel Metallurgical Corporation of North Chicago, the availability of tantalum for civilian surgeons is assured at an early date.

Tantalum has assisted surgeons to return to active life many cases which in the last war would have been disfigured and incapacitated for life. Lost portions of the skull, ears, noses and other parts of the face are being replaced with tantalum. One veteran has a tantalum "belly wall". Nerves which control motion in arms and legs are stitched with tantalum thread and protected while healing with tantalum cuffs. Facial paralysis is relieved by small, saddle-shaped pieces of tantalum and wire used to pull the corners of the mouth to a normal position. This stops the unpleasant drooling and facial distortion which go with the condition. Cleft palates also are being corrected.

ECTOPIC PREGNANCY*

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Ectopic pregnancy is a gestation which occurs outside the cavity of the uterus. It may be located in the broad ligament, the peritoneal cavity, the ovary, or any portion of the fallopian tube, including the interstitial portion. The successful treatment of this condition depends to a large degree upon an early and correct diagnosis.

In 1940 we reported[1] some observations on one hundred and fifty consecutive ectopic pregnancies. Today we wish to report sixty-two consecutive cases of ectopic pregnancy treated in The Medical College of Virginia Hospitals during the past three years: 1940, 1941, 1942. These patients have been divided into two groups, according to a new classification which resulted from an analysis of our original report. The first group was admitted with symptoms of an acute rupture of the ectopic gestation. The second group was admitted with low abdominal pain and a pelvic mass, usually unilateral, resulting from a slow hemorrhage or hemorrhage confined within the folds of the broad ligament.

The difficulty one encounters in making a correct diagnosis on these patients is illustrated by the fact that the cases we are reporting were referred to the hospital for admission to the obstetrical, general surgical, genito-urinary, and medical services.

Eighteen patients (29 per cent) were white, and forty-four (71 per cent) were negroes. Thirty-seven (59 per cent) of the cases gave evidence of sudden or acute rupture, and twenty-five (41 per cent) were classified as old ruptures with definite pelvic masses.

PAIN. All of the patients complained of some type of low abdominal or pelvic pain. It was described as cramp-like in 46 per cent of the acute group, and in 68 per cent of the patients with a palpable pelvic mass. Sharp pain followed by fainting was encountered in 38 per cent of the acute cases and in 8 per cent of the patients with a pelvic mass. In the acute group sharp pain, with or without fainting, was complained of by 73 per cent of the patients.

VAGINAL BLEEDING. In the patients with a pelvic mass, vaginal bleeding was observed in 84 per cent of the cases, and in the recent ruptures it occurred in only 65 per cent of the patients. Profuse vaginal bleeding was not observed in either group. Only one patient passed a blood clot larger than a walnut, and she had uterine fibroids and evidence of old, pelvic inflammatory disease. The absence of excessive vaginal bleeding and large clots is an important diagnostic point in ruling out the abortion cases. We have seen two patients[2] with ectopic gestations in whom the pregnancies continued to grow and remain viable after vaginal bleeding had been observed for several days.

BLOOD PRESSURE. Only one patient in the hematoma group had on admission a systolic blood pressure below 100 mm. mercury, but 24 per cent of the acute group had a blood pressure below 100 mm. systolic on admission. In a different and larger group of cases we found that only 17 per cent of the patients with ectopic gestation presented the classical picture of sudden severe pain, followed by syncope.

PULSE. In the entire series the pulse was above 80 per minute in all except two patients. It was above 90 in 75 per cent of the patients with hematoma. This corresponds with the findings in our first report, in which we noted that the pulse rate was accelerated out of proportion to the elevation of the temperature in patients with ectopic gestation.

TEMPERATURE. In cases with an acute rupture the temperature was below 100F. in 78 per cent of the group, and the temperature remained below 101F. in all of this group. In the hematoma group there were only two patients with a temperature above 101F., and 68 per cent had a temperature which remained below 100F. A slight elevation of temperature is encountered frequently in patients with ruptured ectopic pregnancy of several hours duration.

*Read before the Seaboard Medical Association of Virginia and North Carolina at its annual meeting in Richmond, December 2, 1943.

RED CELL COUNT. Forty-eight per cent of the patients with acute rupture had a red cell count below three million, but only 16 per cent of the hematoma group had such a low cell count.

HEMOGLOBIN. There was a striking comparison in the hemoglobin readings between the acutely ruptured and the hematoma groups. The hemoglobin was below 60 per cent in 59 per cent of the acute group, but below 60 per cent in only 20 per cent of the latter.

WHITE CELL COUNT. Sudden hemorrhage causes leucocytosis, and in the acute ruptures the total leucocyte count was above ten thousand in 70 per cent of the patients, but in the hematoma group it was above ten thousand in only 32 per cent of the cases.

PELVIC EXAMINATION. Pelvic examination should always be done gently, and when pregnancy is present, unnecessary manipulation becomes extremely dangerous. In the presence of ectopic gestation manipulation of the cervix usually causes severe pain. The presence or absence of a palpable unilateral pelvic mass depends upon the duration of the pregnancy, and the amount of hemorrhage or secondary reaction associated with the hemorrhage.

We wish to report in detail on one patient in this series because she had an intra-uterine pregnancy and a ruptured tubal pregnancy at the same time. Following removal of the ectopic gestation she went to term, and seven months after the salpingectomy she was delivered of a full term intra-uterine pregnancy.

L. T. C., a white female, age 30 years, gravida 2, para 1, was admitted to The Medical College Hospital on March 17, 1942, at 9:15 P. M. Her chief complaint was low abdominal pain of two weeks duration, more severe during the afternoon before admission. The last regular menses occurred January 5, two months before admission to the hospital.

History: The patient had one pregnancy four years ago, delivered by classical cesarean section after twelve hours of labor. The convalescense was normal. The operation was performed in another state. She complained of chronic constipation; otherwise the history was negative.

Examination: The blood pressure was, systolic 100, diastolic 60; pulse 80; temperature 99.6F. Urinalysis was normal. Red cell count was 2,620,000; hemoglobin 52 per cent; white cell count 7,900. Pelvic examination: The cervix was soft and the canal closed; fundus of uterus was anterior, soft, and enlarged to the size of a six weeks pregnancy. Admission Diagnosis: Intra-uterine pregnancy, six weeks; adhesion due to former abdominal operation.

The patient continued to have colicky, cramp-like pains in the right lower quadrant, and on March 21 she had severe, low abdominal pain and a small amount of blood-tinged fluid in the vagina. Pelvic examination revealed the uterus to be anterior, soft, and enlarged to the size of a six weeks pregnancy. There was an indistinct soft mass, the size of an orange, in the right side of the pelvis. This was diagnosed as an ectopic pregnancy.

Operation revealed a ruptured ectopic pregnancy, located in the middle third of the right fallopian tube. There was a hematoma, and pelvic adhesions were rather dense. It was necessary to remove the right tube and ovary. The patient's convalescence was normal and her temperature never went above 100F.

Two weeks later her uterus was enlarging normally, and her convalescence had remained uneventful. She was watched closely, and on October 25, 1942, seven months after the ectopic gestation was removed, she was delivered by cesarean section of a living female child which weighed six pounds and nine ounces. Thirteen days after operation the mother and baby left the hospital in good condition.

The treatment for ectopic gestation is immediate operation. If the patient has lost any appreciable amount of blood, transfusion with whole blood should be started at the time of operation or as soon thereafter as possible. If blood or plasma is not available, intravenous fluids should be given during the operation.

Most of the patients with acutely ruptured ectopic pregnancies, in this report, were given transfusions during the operation or soon thereafter. Only 6 patients, or 25 per cent of those with pelvic masses, needed immediate transfusion.

SUMMARY AND CONCLUSIONS

Sixty-two consecutive cases of ectopic pregnancy with no deaths have been reported.

The importance of a careful and accurate history has been emphasized. The frequent error of considering abnormal or irregular vaginal bleeding as a delayed menstrual period has been discussed.

The pain in this series of cases was cramp-like in 46 per cent of the patients with an acute rup-

ture, and 68 per cent of the patients with a palpable pelvic mass.

The textbook picture of ruptured ectopic pregnancy, with sudden severe low abdominal pain followed quickly by syncope and shock, was observed in less than 20 per cent of the patients in this series.

Slight elevation of temperature, rarely above 101F., is very characteristic of ruptured ectopic gestation.

A leucocyte count above ten thousand is rarely encountered after the rupture has existed long enough for a distinct mass to form. Leucocytosis is frequently seen after an acute rupture of an ectopic gestation.

The importance of differentiating between partial rupture of an ectopic pregnancy with prolonged symptoms and acute rupture has been discussed.

A case of ruptured tubal pregnancy, removed at operation, with continuance to term of a co-existing intra-uterine pregnancy, has been reported.

The treatment for ectopic gestation is immediate operation to control or prevent hemorrhage. Transfusions with whole blood and other supportive measures should be carried out during the operation if there is evidence of hemorrhage or shock.

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Physician Population of the U. S. Increased by 2,570 Last Year.

There were 5,952 additions to the medical profession in 1943, according to the data presented in the forty-second annual compilation of medical licensure and allied statistics by the Council on Medical Education and Hospitals of the American Medical Association and published in *The Journal* of the Association for May 13.

The report says that the number of physicians removed by death in 1943 was 3,382. "It would appear, therefore," the report says, "that the physician population in the United States last year was increased by 2,570. In view of the accelerated curriculum with two classes graduating from most schools in 1943, one might expect that additions to the profession should be considerably higher. This in reality is the case at the present time. However, many physicians who obtained M.D. degrees in December of 1943 were not able to receive licenses until early in the year 1944, owing to administrative details. . . .

"Estimated figures indicate that on February 1, 1944 the number of physicians in continental United States, including those licensed in 1943, was 186,496. Excluding physicians who are in military service, engaged in full time hospital work, retired, not in practice or engaged in full time teaching, there remain about 100,000 physicians in private practice, some of whom are part time teachers. . . ."

Throughout 1943, 8,392 graduates were examined for licensure, of whom 7,478 passed and 914 failed. Of 6,427 graduates of approved medical schools in the United States only 1.5 per cent failed. Of 76 graduates of approved Canadian schools, 15.7 per cent failed; of 101 who graduated from approved schools no longer operating, 5.0 per cent failed; of 1,031 graduates of faculties of medicine located in countries other than the United States and Canada, 49.8 per cent failed. There were 38.4 per cent failures among 757 graduates of unapproved schools.

Of particular interest is that portion of the report concerning licensure for relocated physicians. The report says that "Removal of physicians from civilian practice has resulted in a shortage . . . in critical areas, especially in some industrial and rural sections of the country. To assist physicians attempting to relocate in such areas, the licensing boards of fifteen states provide for the issuance of temporary permits or certificates to practice medicine. . . ." A total of 244 such temporary permits were granted by the fifteen states during 1943.

DIAGNOSIS AND TREATMENT OF PRIMARY ATYPICAL PNEUMONIA*

VERNON D. OFFUTT, M.D.,
Kinston, North Carolina.

Recently the widespread occurrence in this country of an epidemic form of nonbacterial pneumonia, described variously as acute influenzal pneumonitis, disseminated focal pneumonia and virus pneumonia has renewed our interest in this respiratory disease.

The essential clinical characteristics of primary atypical pneumonia have been described in a number of publications over the past nine years. Allen, in 1935, analyzed and reported on 50 cases at Fort Sam Houston which conformed essentially to those seen recently, and used the term acute pneumonitis to designate a form of respiratory infection characterized by a benign course, few physical signs, and x-ray evidence of a localized inflammatory process in the lungs.

In 1938, Reimann reported on eight cases occurring in Philadelphia and made studies to determine the etiological agent. These were all negative for the influenza virus A and B, as well as psittacosis virus which is known to produce similar pulmonary lesions. A filterable infectious agent was recovered from the nasopharynx of one and from the blood of another, but evidence that it was actually the cause of the disease was incomplete. He was, therefore, led to regard it as a separate disease entity pending further study. According to British investigators the probability of obtaining the virus diminishes rapidly after the third day, and they also suggest that when the virus attacks the lungs there is less of it in the upper respiratory tract.

Examinations of the sputum in our cases showed the usual nasopharyngeal and upper bronchial flora, such as streptococci viridans, staphylococci, diphtheroids, gram-negative bacilli, and occasionally pneumococci of the higher types, except in one case tubercule bacilli were found where that disease coexisted.

CLINICAL FEATURES

Reports of this disease indicate that there is a wide variation in the intensity of the symptoms, and the diversity of signs in the severe cases, such as encephalitis to the milder respiratory cases, would suggest that no single etiologic factor is in all instances responsible for the disease.

The essential features of our cases were as follows:

1. With or without upper respiratory symptoms and signs.
2. Repeated chills and fever.
3. Sweating profuse.
4. Non-productive cough.
5. Lack of physical signs first 3 to 5 days.
6. Normal leucocyte count and differential.
7. Normal sedimentation rate.
8. Sputum non-specific.
9. Non-responsive to sulfonamides.
10. X-ray evidence.

The discrepancy between the physical signs and the x-ray is marked and the latter is not always characteristic. Diffuse soft mottling fanning out from the hilum and fading into normal lung is the rule, not as dense as lobar pneumonia and may show involvement of one or all lobes simultaneously or successively. The lesion may advance in one portion of lung while regressing at the original site. Resolution as seen by x-ray generally lags considerably behind clinical improvement.

TREATMENT

The treatment of the disease has been entirely symptomatic and therefore quite unsatisfactory. Adequate fluid intake, codeine sufficient to control cough and medicated steam inhalations appear to have some effect in loosening secretions. The sulfonamides, if used at all, are given in the first days as a diagnostic and therapeutic test, and, if there is no response in thirty-six to forty-eight hours and certainly after the diagnosis is established, the drug is discontinued. There is evidence that it may actually do harm if use is continued or used at all.

Due to this unsatisfactory state of treatment and because of the prolonged period of fatigue which disables some cases for as long as four to six weeks, we have endeavored to find more effective means of treatment. Although the results of x-ray therapy of lobar pneumonia are well known, there is little information available on treatment of atypical pneumonia. Oppenheimer has reported on a series of

*Read before the Seaboard Medical Association of Virginia and North Carolina, at Richmond, Va., November 30-December 2, 1943.

56 cases, none of which had responded to medical treatment, and failure to respond to sulfonamide therapy was considered a diagnostic sign. Forty-five were termed cured in three to five days. The remainder were over fourteen days' duration when first treated. According to inquiries made at several institutions, it has not been used so far in treatment of the virus pneumonias.

Accordingly we made a point to treat all cases of primary atypical pneumonia with x-ray therapy since September and we have thus handled twelve cases to this writing. Our cases received 100 r doses daily or every other day depending on severity and response, alternating front and back or alternating sides if both lungs were involved. None received more than the fourth dose and the majority had three. The temperature began to decline after the first treatment, and usually after the second or third dose responded by rapid lysis. One case which was referred after ten days' treatment for an associated sinus infection responded equally well after the second treatment.

With surprising uniformity fever and symptoms subsided and after one or two treatments most of the patients spent their first comfortable night after days of harassing cough and discomfort. With but one exception all cases were fever free and discharged from the hospital in one week or less. The one exception was complicated by severe diabetes and developed a sterile effusion after treatment which later absorbed and made a normal recovery. No unfavorable reactions occurred in our cases with this therapy. In the presence of leukopenia no further decrease in WBC was observed.

Our investigation is continuing; however, with the cases thus far handled it is our opinion that roentgen therapy is the most effective mode of treatment for control of the cough and fever, and greatly hastens convalescence.

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Kinston Clinic.

A Medical Art Show by Minneapolis Public Library.

The Minneapolis Public Library staged a unique art show June 21-July 22, representing the work of leading medical artists the country over. This is part of a program for extending the services of the Public Library to one of the foremost medical centers in the United States, and dramatizing the resources of such departments as the Art, Technical, and Clipping Service.

A display of rare medical books and prints formed an important part of the exhibit. From the library of the Surgeon-General in Washington were four books showing the earliest examples of anatomical drawings, the datelines varying from 1551 to 1682; from the library of Dr. Shirley Miller of the University of Minnesota, a copy of the 1783 Vesalius, and photostatic copies of early anatomies. A copy of the edition of Vesalius, printed from the 1543 plates by the New York Academy of Medicine, was loaned by Modern Medicine.

Blood Donor Service.

"Since its inception (Feb. 3, 1941) the American Red Cross Blood Donor Service has grown from a single center producing 200 bleedings a week to thirty-five centers delivering up to 120,000. In the same period, the nursing staff has increased from four to over 900,—the largest group of nurses employed by the American Red Cross on any single project."

With this introduction, the article *The Nurse and the Blood Donor Service* in the June, 1944, *American Journal of Nursing* discusses the objectives of a blood donor service and the functions of nurses in attaining them.

Since January 1, 1943, no "military eligible" nurses have been employed by the Blood Donor Service. In view of the nursing shortage, attempts have been made to recruit nurses who were not in active nursing and to employ a proportion of nurses in the upper age brackets.

HEALTH AND GOVERNMENT

FRANK L. APPERLY, M.D., D.Sc.,
Medical College of Virginia,
Richmond, Virginia.

Of those who have written about public health problems and especially of their effects on the people as a whole, too many have concerned themselves with special aspects or small fragments of the problems involved, and too few with the fundamental or grass-root bases on which public health depends. The following is an attempt to determine these fundamentals and to stimulate others to do likewise or to define them more clearly.

One of the aims of a civilized community is the attainment of the maximum of freedom and comfort for the individual together with the minimum of interference with the freedom and comfort of others. However, many of the conditions necessary to attain this end cannot be provided by the individual alone, but only by the people acting as a whole, through their representatives and servants, i.e., by government.

One of the greatest factors concerned in the attainment of these aims of civilization is the health of the people. But health is a most complex state, depending on many conditions. Among these conditions are:

A. Certain physical requirements—
   (a) Nutritional standards (involving national and international arrangements, treaties, trade, etc.).
   (b) Facilities for exercise and leisure.
   (c) Physical environment (housing, sunshine, water supply, disposal of sewage, etc.).

B. Certain psychic or "spiritual" conditions—
   (a) Social environment.
   (b) Security from fear and want.
   (c) Educational facilities.
   (d) The "will to health."

These conditions in turn depend ultimately on the people themselves, acting through government, whose function is to plan, regulate and coordinate the activities of the people so that these conditions shall be fulfilled. Some of these conditions, of course, pre-suppose medical knowledge and an efficient medical service, which in turn depend upon:

(a) Medical education—undergraduate and post-graduate.
(b) Available medical and scientific knowledge (e.g. libraries).
(c) Medical research.
(d) Absence of economic barriers to the utilization and growth of the above.

All of these again depend upon government, public and private endowment.

From this it would seem that the conditions governing the health of the people depend ultimately upon government, whether Federal, State, or local, since only these agencies can coordinate, and to some extent create, the various conditions enumerated.

When, however, the health of the individual is impaired, it would seem that in such a personal matter he should be free to consult whomsoever he wishes. Even here he is again regulated (and properly) government which provides:

(a) That if his disease is such as to endanger his neighbors, he is a public health problem.
(b) That his consultant is one who has had the requisite training, experience and the right to practice his profession.
(c) The personnel and equipment when the sick person is a member of the armed or essential services, or for reasons of poverty cannot obtain or afford medical attention and is therefore liable to become a danger or a burden to the people. The matter is then one of the general concern. For this reason it is a function of government to see that medical services are available to all, regardless of economic status.

Only when these conditions are fulfilled is a man's illness no longer a public affair, but his own private business. He should therefore be free to consult an expert of his own choice, in exactly the same way as he is free to choose his own lawyer, engineer, or architect, his choice being limited only by distance and by his purse. This is properly the field of private practice.

In conclusion, this brief outline is put forward in an attempt to define clearly the basis or foundation on which plans involving government and medicine must be built. If this basis is accepted, it becomes our duty to educate ourselves, the public and government concerning those matters which pertain to government and those which are properly private and therefore free from governmental interference.

CASE REPORT OF MATERNAL DEATH

MATERNAL HEALTH COMMITTEE,
MEDICAL SOCIETY OF VIRGINIA

A 36 year old multipara (Para VII) consulted her physician several times during her pregnancy and her pregnancy was relatively normal. The doctor had delivered some of her previous children, and all the labors had been normal. The patient started in labor at 6 P. M. on March 10. The doctor saw her soon thereafter and gave her 1 cc. of thy-tuitary. He left after examining her and returned at 10 P. M. At this time the membranes were ruptured artificially and 1 cc. of pituitrin was given. The pituitrin (1 cc.) was repeated in one hour. The patient had not made much progress by midnight so she was allowed to rest until the following morning. She was seen again at 6 A. M., March 11. There had been a few pains during the night but the patient had made little progress. The cervix was two or three inches dilated at this time. Forceps were applied under light chloroform anesthesia and an unsuccessful attempt was made to bring down the head. About 10 A. M. a consultant was called and the consultant advised giving the patient more time. Nevertheless, an attempt at forceps delivery was made this time under ether anesthesia. At noon the forceps were again applied, this time without an anesthetic, and again the forceps slipped off. During the day she received 6 grains of sodium amytal and four doses of 8 or 10 minims of pituitrin. At 6 P. M., twenty-four hours after the onset of labor, she was again seen by the consultant. Her labor pains had been irregular and of poor quality. The patient was exhausted and did not look so good. It was therefore decided to send her to the hospital.

She arrived at the hospital at 2 A. M. on March 12, moribund, in shock, pulseless, and cold, temperature 101, pulse 130, respirations 50. There was a pregnancy near term. The vulva was badly traumatized. Her condition was too bad to attempt delivery. The patient was put to bed and external heat applied. In addition 10 per cent glucose in normal saline was given by venoclysis and cardio-respiratory stimulants were administered. The patient died undelivered about 7 hours after admission to the hospital.

It was the opinion of the Committee that this patient had a ruptured uterus, although there was no autopsy to confirm this. Whether the rupture was caused by the oxytocics used or by the attempts at forceps delivery, one cannot say. Either could have caused it, and both were reprehensible. In the history of the case there appears no indication for doing anything to this patient while she was in her home. Whether she could have been saved after she entered the hospital by a blood transfusion and a laparotomy is a question.

The lessons to be learned from this case are two: First, pituitrin is a dangerous therapeutic agent that should rarely be used before the delivery of the baby. If it is used in antepartum, the dose should be extremely small and the patient should be watched for a tetanic contraction. Should this occur, an anesthetic should be administered at once; Second, the use of forceps is contraindicated before the cervix is fully dilated.

Make your reservations for the Medical Society of Virginia Meeting in Richmond, October 23-25.

PUBLIC HEALTH

I. C. RIGGIN, M.D.,
State Health Commissioner of Virginia

The report of the Bureau of Communicable Diseases of the State Department of Health for June, 1944, as compared with the same month in 1943, and for the period of January through June, 1944, compared with the same period in 1943, follows:

	JUNE 1944	JUNE 1943	JAN.-JUNE 1944	JAN.-JUNE 1943
Typhoid and Paratyphoid Fever	11	19	55	70
Diarrhea and Dysentery	640	326	1537	928
Measles	949	1016	16658	8868
Scarlet Fever	103	64	1851	1033
Diphtheria	13	11	116	163
Poliomyelitis	8	3	14	15
Meningitis	24	50	395	640
Undulant Fever	8	4	25	14
Rocky Mountain Spotted Fever	14	8	21	9
Tularemia	7	5	27	29

MORTALITY AMONG SCHOOL CHILDREN

The mortality rate for children of school age (5 to 14 years) is lower than for any other age-group. The low and steadily declining rate, however, may me a misleading health index, as the disabling and crippling diseases which attack children show their effects in the mortality rates of young adults.

Despite the declining rate, deaths of children 5 to 14 years of age in the State last year totaled 510, with a rate of 0.9 per 1,000 population. Twenty years ago, the rate was 1.9, with 1,084 deaths—more than twice the number occurring in the past year.

As a result of the success in the control of communicable and other childhood diseases, the most important cause of death numerically, at the present time, among school children is accidents of all types. There were 99 deaths due to accidents in Virginia in 1943, including falls, burns, drowning, injury by agricultural machinery, and other types. Motor vehicles took an additional death toll of sixty-one children. Although increasing motor-vehicle traffic during the past two decades has been a great menace to the safety of school children, educational safety programs are proving effective in significantly reducing the death rate for accidents of this type.

Pneumonia and influenza, twenty years ago the leading cause of death among children 5 to 14 years of age had dropped, by 1943, to second place, with 52 deaths. In recent years, the result of advances in pneumonia therapy is seen in the greatly accelerated rate of decline in mortality from this cause.

Although the tuberculosis death rate has decreased so significantly in the past two decades, this disease ranked third among the leading causes of death among school children in the State in 1943. There were 39 deaths attributed to this cause within this age-group.

The fourth important cause of death among children 5 to 14 years was heart disease, with 32 deaths. Of this number 25, or 78 per cent were specified as rheumatic. *Mortality* from rheumatic fever, however, does not begin to indicate the prevalence of this disease among school children because of low fatality rates for the first attack. Due to the high incidence and serious after-effects in later years, this cause of illness and death becomes one of the major health problems of the present day.

Nephritis, as a cause of death among school children, came fifth in rank last year, causing 17 deaths.

In spite of the fact that appendicitis held the same position (sixth) among the leading causes of death in 1943 as it did in 1923 for the 5 to 14 age-group, there was a 68 per cent reduction in mortality during the period. Fourteen deaths resulted last year from this cause in comparison with 44 deaths twenty years ago.

Death rates for boys of school age were consistently higher than those for girls. The ratio of the male rate to the female in the State in 1943 was 1.33.

WOMAN'S AUXILIARY
to the
MEDICAL SOCIETY OF VIRGINIA

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To the Wives of the Young Doctors.

Many of the founders of the Woman's Auxiliary have already gone on to the great beyond. Those of us who joined the family of doctors' wives a little later than the founders are fast approaching the age of incapacity and the future of the Auxiliary lies in the hands of the wives of the young doctors.

You may not be especially interested; perhaps you have not given it much thought. I'm sure many of you have said that the projects of the Auxiliary are also projects of your other clubs—Parent-Teacher Associations, Woman's Clubs, etc., and you feel that duplication of effort is unnecessary and a loss of time. This is true in part, but would you like to turn over to these organizations the entertainments of the medical societies, the aiding of doctors' families in distress and the requests of your local medical society for services pertaining to their specific needs?

There is something clannish about the Auxiliary. No wives of other professions belong, as in other clubs, and we all have the same interests at heart—the success of our particular doctor, the dignity of the profession, the rights of the individual physician against the inroads of State Medicine and many other common interests. With such singleness of purpose should we not band together and strengthen our purpose by unity?

Most of you have been or will be asked to serve the Auxiliary, local or state, in some capacity. Perhaps you have neither the time nor the inclination and it takes both. Even if you accept you will probably ask yourself why you did it and wish you hadn't. But be patient and put your best into your office or chairmanship, whichever it may be, for the term will not be long and when it is over you will be glad that you met this responsibility. And your husband, even though he may be one of those who is not yet enthusiastic about the Auxiliary, will feel a secret satisfaction and pride that his wife had a part in carrying on the work.

RUTH PENDLETON HARRISON WILSON,
(MRS. FRANKLIN D.)
Past President, 1937-38.

Leigh-Hodges-Wright Memorial Bed Fund.

In 1936 a resolution was adopted by the Woman's Auxiliary to the Medical Society of Virginia to assume the expense of supporting an "Auxiliary Bed", in one of our state tuberculosis sanatoria, to be used by a physician or some member of a doctor's family. The members of the Advisory Council were much interested in this project and the matter was taken up with those in charge of the sanatoria at Blue Ridge and Catawba, who thought a real service could be rendered by such a plan.

During the first months after the adoption of this plan, the fund was used to assist the daughter of a retired physician, who was then a patient at Blue Ridge. After she had been discharged, we were asked by Dr. Brown to consider a young man who had to enter the sanatorium just one month before getting his degree in medicine at the University of Virginia. The Auxiliary found much real satisfaction and pleasure in being able to render assistance to this young man for a number of years.

In 1938, it was voted to adopt the name, "Leigh-Hodges-Wright Memorial Bed", in honor of the memory of these three physicians who had all been members of the State Advisory Committee.

At the present time, the fund is not being used, though it is available at any time there should be a call for it. Last year it was decided that an effort be made to build this fund up to a worth-while amount with the hope that it may some day become an endowment.

It is a known fact that war always increases the spread of tuberculosis, and it is the hope of the committee that we will be prepared to meet an emergency at any time, should such occur.

ANNE SEAY WRIGHT,
(MRS. FLETCHER J. WRIGHT)
Chairman, Leigh-Hodges-Wright Memorial Bed Fund, and Past President of the Woman's Auxiliary to the Medical Society of Virginia.

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The Treatment of Eclampsia

ECLAMPSIA can be prevented easily by ordinary prenatal care. Nothing in medicine yields such large dividends in health and happiness as the medical supervision of a patient during pregnancy. On the other hand, when this supervision is omitted, a considerable number of women will develop toxemia of pregnancy. This is as certain as taxes and death. In the South where prenatal care is greatly neglected, toxemia of pregnancy is the most frequent cause of maternal death. The treatment of eclampsia which is the terrible climax of untreated toxemia is the most neglected field in medicine.

There are several causes for this neglect. In the first place, the disease is so easily prevented that one has the feeling, subconscious in most instances, that the patient does not deserve the painstaking and troublesome treatment that these patients must have for the best results. In the second place, the disease has become so rare in the teaching centers that the medical students are not taught how to treat it. In fact, the professors of obstetrics themselves seem not to know the best treatment. This was brought out at the recent meeting of the American Gynecological Society. There were two papers on this subject on the program. The first was entitled "The Failure of the Conservative Treatment of Eclampsia", and the second, "Management of Eclampsia; Conclusions Based on 142 Consecutive Cases in a Five-Year Period without Maternal Death". These two papers covered a five-year period, one beginning January 1, 1938, and the other January 1, 1939. This was the only thing that the papers had in common except the continuous administration of oxygen through a nasal tube. The first paper dealt with 51 eclamptics, 37 of which were admitted as emergencies, not having been seen previously by any member of the hospital staff. There were 12 maternal deaths and 18 infant deaths, 23.5 per cent and 35 per cent, respectively. In the 14 patients who received prenatal care in the clinic, there was one fetal death and one maternal death. The latter was treated by cesarean section after she had had one convulsion. She died

8 hours after the operation without showing any signs of responding to the treatment. Thirteen of the remaining 37 cases were classified as severe by Dieckman's criteria. Eleven of these 13 mothers died as did 9 of the 18 infants. The general plan of therapy was "one of conservative medical management in an attempt to control the convulsions and establish an increasing urinary output". For this purpose the author used sodium amytal, supplemented by the administration of 100 cc. of a 25 per cent solution of magnesium sulphate intramuscularly. Twenty per cent glucose solution was given intravenously in amounts of 1,000 to 1,500 cc., two or three times each 24 hours. Continuous oxygen by nasal catheter was given to most patients. The results in the treatment of the severe eclamptics were so poor that this author concludes that it would be better to treat this group by early cesarean section, although apparently he has had no experience to support this claim. He says that the result so far as the mother is concerned could be no worse, and that more babies would be saved. In contrast to this, the second author reported 142 consecutive cases, 36.6 per cent of which were severe by Dieckman's classification, without a maternal death. There was a gross fetal mortality of 24.2 per cent. The conservative treatment that produced such remarkable results consisted of constant and continuous supervision of the patient not only while she was having convulsions but also until she was out of danger. The patient was put to bed in a quiet, darkened room and false teeth, rings, and bracelets were removed. Side boards were used to protect the patient against falls and oxygen was given continuously by nasal tube. Medical and obstetric examinations were deferred until sedation had become effective. Morphine sulphate, gr. ¼ and sodium phenobarbital grs. 5 were given hypodermically at once. Fifteen minutes later, if convulsions were still present or if hyperirritability were extreme, 20 cc. of a 10 per cent solution of magnesium sulphate were given intravenously. Sodium amytal was given intravenously in doses of 3 to 6 grains or the morphine was repeated, but the magnesium sulphate was not repeated unless convulsions recurred. The sedatives were used in the smallest possible doses to control the hyperirritability. Five hundred cc. of a 20 per cent dextrose solution were given by intravenous infusion over a period of about fifty minutes. If there were evidence of cardiac failure or pulmonary edema, only 50 to 150 cc. of a 50 per cent solution were given. On the other hand, if there were evidence of dehydration, the patient was given 1,000 cc. of a 5 or 10 per cent solution. The intravenous administration of glucose was discontinued as soon as the patient was able to take sufficient fluids by mouth. Persistent oliguria or anuria associated with such conditions as anemia, hypoproteinemia or hypotension demanded special additional measures, (blood transfusions, plasma transfusions, or diathermy over the kidney region). Twelve patients with marked cerebral irritation were treated by spinal drainage. Venesection was not used. "Although in the presence of acute cardiac failure, drugs of the digitalis group are theoretically of at least temporary value, we have found them of no particular benefit in the few cases in which we have used them." The vasodilator drugs such as veratrum viride, the nitrites, and nitroglycerine were not used.

The second author stressed the importance of continuing the constant treatment during the postconvulsive stage. The continuous oxygen is kept up, and the air-passages are kept free from mucous and other material. The head of the bed is still further elevated and the height is changed at frequent intervals. Blood pressure and urinary output are determined at hourly intervals at first and at four hour intervals later. An obstetrical examination is now made and a large dose of magnesium sulphate is given by mouth. Sedation is used in quantities just sufficient to control the cerebral irritation. The patient is encouraged to drink fluids, (sweetened fruit juices, water, coca-cola, milk, coffee, tea or beer). A period of watchful expectancy follows, during which

time the patient is fed a high protein, low salt diet. If the improvement of the patient continues, pregnancy is not interrupted until she has become normal. Medical induction is then started. It may have to be repeated several times. He reserves amniotomy for those cases that do not respond to medical induction. In one instance a cesarean section was done. Operative delivery was held to a minimum. Cesarean section was performed only nine times in the entire series. Local analgesia was used for all varieties of operative work. Nevertheless, in the 104 cases of antepartum and intrapartum eclampsia, there were 3 instances of peripheral vascular collapse, 4 instances of postpartum hemorrhage, 4 instances of persistent anuria, and 4 instances of profound coma. He recommends that 500 cc. of a 20 per cent glucose solution be started as soon as the patient is prepared for delivery, and that it be given very slowly. This measure aids in preventing shock, and obviates the difficulty of entrance into veins if vascular collapse should occur. Morphine is administered as soon as delivery is completed.

Privately, the professors expressed a doubt that 142 consecutive cases of eclampsia could be treated without a maternal death. Our own experience in the past 19 years would indicate that such a result is not impossible. If we eliminate epilepsy, as did the second author, we have a series of 147 consecutive cases of bona-fide eclampsia with 6 maternal deaths. In the treatment of these patients our guiding principles have been: (1) Stopping the convulsions with intravenous magnesium sulphate solution, sodium amytal or veratrum viride, (2) Good nursing care with emphasis on rest, (3) Digitalization of the patient early as a prophylaxis of pulmonary edema, and (4) Fluids by way of the gastro-intestinal tract. Intravenous fluids were given only when oliguria was developing. Purgatives, enematas and lavages were omitted so that the patient could have much needed rest. Between May, 1929, and June, 1943, we had no maternal deaths. Our streak of good luck was broken by our being called in consultation to see a patient in another city, arriving just before the patient died. This patient had received good prenatal care and had showed no signs of toxemia. She developed fulminating eclampsia four and three-quarter hours after a breech delivery under chloroform anesthesia. Her treatment was interesting but hardly conformed to the objectives outlined above. She was treated with epsom salts, calomel, jalap, croton oil, and colonic irrigations. When she had a vascular collapse with pulmonary edema, she was put in an oxygen tent and given digifolin. She did not regain consciousness. Bryant and Fleming who rely upon veratrum viride, intramuscular injections of magnesium sulphate, and 500 cc. of a 20 per cent solution of glucose, and no sedatives to control convulsions, had a series of 140 consecutive cases with 2 maternal deaths, and these were due to infection which followed the induction of labor with bags. Arnell, the second author, emphasizes personal care of the patient so that she gets just the amount of treatment that her case demands and no more, and that seems to be the crux of the situation.

Words We Never Knew Till Now

AFTER a year's lapse the American Medical Association resumed its annual intensive post-graduate course with an attendance of something over 7,000. The sections had capacity audiences. In some there was standing room only. The exhibits also were well attended. They covered the widest range of subjects from how to do a venipuncture to mosquito control in the tropics. The exhibitors talked to constantly assembling groups of avid listeners.

Your editor was impressed among other things with the number of new terms, and

old ones with new uses with which he met. As was to be expected, the nervous and mental group headed the list. He stopped in at their meeting for a moment and was greeted with "indigestible toxic substances of the mind". Just what this is he has yet to learn. The bacteriologists have a way of renaming their pets just as the ordinary practitioner gets used to the old names. Food poisoning is no longer caused by the paratyphoid group of organisms but by the *Salmonella B., paratyphoid A.* or a kindred organism. Of course, that is not really new for they have been talking of the Salmonella for several years, but it is hard for an old mind to get used to the new terms. Now they talk of the *Shigella* and the person affected does not have dysentery, but *Shigelosis. Muma* is the name that is suggested for the acute form of filariasis, and when the patient has the larvae in the blood stream, he has *filaremia.* This brings to mind that live trichina larvae can be demonstrated on the screen by a *microvivarium.* We heard for the first time of *penicillin-fast bacteria* and we hope sincerely that this proves to be a figment of the imagination.

The renewed interest in vascular disease has developed a crop of new terms, such as *arterial confusion, local shock,* and *Homas's sign.* We had heard of the latter before we went to Chicago, but the other two are brand new to us. Then we have a note on *Zudock's syndrome,* but for the life of us we do not know now what it is. We heard again about the *female prostate* that gives such baffling and elusive symptoms in elderly ladies. We learned also that normally a woman towards the end of pregnancy develops a number of *microkaryocytes* in her bone marrow.

One who has passed the American Board of This or That is now spoken of as a *Certificatee.* No doubt we missed a lot of new words although we worked pretty hard from morning to night. The convention was so big that it was hopeless for one to try to cover it adequately. There was one extra-mural word that we learned from the Chicago papers that was quite disturbing. There was an editorial comment on the wording of the Supreme Court's decision that it is unconstitutional to deprive a voter of the privilege of voting for his *rulers.* We were brought up on the doctrine of the sovereignty of the people, and that those we elected to high offices were the servants of the people. At least that is what they used to tell us. This change in mental attitude in Washington is alarming. But in this changing world we must be surprised at nothing.

Floral Eponym (18)

BIGELOVIA

Jacob Bigelow, 1787-1879

BIGELOVIA comprises more than thirty species of western American herbs or low shrubs. The one originally described closely resembles a goldenrod to which Bigelovia is very closely related. Dr. Jacob Bigelow, the first of the celebrated Bostonian medical family, was born in Watertown, Massachusetts, the son of a clergyman. He graduated at Harvard, and then went to the University of Pennsylvania where he received his medical degree in 1810. Returning to Boston, he became the partner of Dr. James Jackson. In middle life he was visiting physician to the Massachusetts General Hospital and Professor of Materia Medica at Harvard. He was one of the first to insist on the self-limited character of disease and worked for a reform of medical education. Music, modelling, and botany were his refreshment. He wrote Florula Bostoniensis, and Medical Botany of the United States. The latter is a three volume work, beautifully illustrated, which is now quite rare.

Societies

Northampton County Medical Society.

This Society, at a recent meeting, elected delegate and alternate to the State meeting to be held in Richmond in the Fall, and voted to discontinue participation in the medical insurance scheme for borrowers from the Farm Security Administration.

Dr. J. M. Lynch of Cape Charles and Dr. W. C. Henderson of Nassawadox are president and secretary, respectively, of this Society.

The Patrick-Henry Medical Society

Held its regular quarterly dinner meeting in Martinsville on July 7. After a short business meeting a very interesting talk followed by discussion was given and led by Dr. Roger H. DuBose of Roanoke. His subject was "Fluids in Children".

Dr. R. H. Walker is president of this Society and Dr. T. H. Dickerson, secretary. Both are of Martinsville.

Fauquier County Medical Society.

At a meeting of this Society on July 13, the following officers were all re-elected for the coming year: President, Dr. Stewart McBryde, Manassas; vice-presidents, Drs. George H. Davis, Warrenton, and Wade C. Payne, Haymarket; and secretary-treasurer, Dr. J. Frank Folk, Warrenton. Delegate and alternate to the State Society meeting were also named.

News

Re-appointed State Health Commissioner.

Governor Darden has announced the re-appointment of Dr. I. C. Riggin as State Health Commissioner of Virginia for another term of four years, effective July 1. Dr. Riggin has been serving in this position since July, 1937, when he was named to succeed Dr. Warren F. Draper who was recalled to service with the U. S. Public Health Service after having been loaned to this State.

Superintendent at State Colony.

Dr. Carl W. White has been appointed superintendent of Lynchburg State Colony, succeeding Dr. G. B. Arnold. Dr. A. D. Hutton, who has for several months been acting superintendent, has returned to Marion. Dr. White is a native of Danville and received his medical education at Jefferson Medical College, Philadelphia, and was recently an assistant physician at Rochester State Hospital, in Minnesota.

News from the University of Virginia.

On Tuesday, May 16, Dr. W. W. Waddell gave a lecture before the West Virginia State Medical Association, meeting in Wheeling, on the subject, "Recent Improvements in the Treatment of Purulent Meningitis".

Dr. Dudley C. Smith attended meetings of the Society for Investigative Dermatology, American Dermatological Association, and American Medical Association in Chicago, June 12 to June 22. He was elected Vice-Chairman of the Section on Dermatology and Syphilology.

Dr. Samuel A. Vest presented a paper at the meeting of the American Medical Association in Chicago in June on "An Inspection Lens-Sheath as an Aid to Transurethal Resection".

At the meeting of the American Urological Association in St. Louis in June, Dr. Vest spoke on the subject "Should Nephroureterectomy Be the Routine Procedure in Tumors of the Ureter?" and conducted an exhibit on conservative surgery of the ureter.

Dr. Vincent W. Archer, Professor of Roentgenology, served as chairman of the Committee on Awards, Scientific Exhibits, during the meeting of the American Medical Association in Chicago, June 13 to 16.

American Medical Association.

Dr. Herman L. Kretschmer, Chicago, succeeded Dr. James E. Paullin, Atlanta, Ga., to the presidency at the annual meeting of the Association held in Chicago, June 13-16. Dr. Roger Irving Lee, Boston, was named president-elect.

There was a registered attendance of 7,284. The following attended from Virginia:

Dr. Mallory S. Andrews, Norfolk.
Dr. Vincent W. Archer, University.
Dr. W. A. Barker, Roanoke.
Dr. Arthur S. Brinkley, Richmond.
Dr. W. E. Butler, Norfolk.
Dr. W. H. Copley, Richmond.
Dr. W. Gayle Crutchfield, University.
Dr. Donald S. Daniel, Richmond.
Dr. J. Wyatt Davis, Jr., Lynchburg.
Dr. J. H. Deyerle, Harrisonburg.
Dr. W. K. Dix, Richmond.
Col. M. F. DuFrenne, Ft. Myer.
Dr. W. C. Elliott, Lebanon.
Dr. Joseph E. Feingold, Roanoke.
Major Gregory G. Floridis, Camp Pickett.
Dr. R. W. Fowlkes, Richmond.
Dr. Emily Gardner, Richmond.
Dr. John J. Giesen, Radford.
Dr. Hugh Griffin, Clinchco.
Dr. R. Bryan Grinnan, Norfolk.
Dr. Harvey B. Haag, Richmond.
Dr. Edwin A. Harper, Lynchburg.
Dr. E. C. Harper, Richmond.
Dr. C. C. Hatfield, North Holston.
Lt. Col. Edmund Horgan, Delaplane.
Dr. J. Shelton Horsley, Richmond.
Dr. William S. Hotchkiss, Norfolk.
Dr. K. W. Howard, Portsmouth.
Dr. J. Morrison Hutcheson, Richmond.
Dr. Jeannette M. Jarman, Hot Springs.
Dr. M. B. Jarman, Hot Springs.
Dr. E. S. Jones, Hampton.
Dr. Frank A. Kearney, Hampton.
Dr. Linwood D. Keyser, Roanoke.
Dr. Edward W. Kunckel, Richmond.
Dr. J. W. Love, Alexandria.
Dr. Eugene L. Lowenberg, Norfolk.
Dr. Walter B. Martin, Norfolk.
Dr. W. Ambrose McGee, Richmond.
Dr. Carl W. Meador, Richmond.
Dr. E. B. Mewborne, Newport News.
Dr. H. B. Mulholland, University.
Dr. Thos. W. Murrell, Richmond.
Dr. B. W. Nash, Timberville.
Dr. R. B. Newman, Newport News.
Dr. B. L. Parrish, Norfolk.
Major E. A. Pushkin, Camp Pickett.
Dr. Samuel B. Pope, Norfolk.
Dr. Wm. R. Pretlow, Warrenton.
Dr. I. C. Riggin, Richmond.
Dr. H. L. Riley, Jr., Lynchburg.
Dr. M. P. Rucker, Richmond.
Dr. J. W. Sayre, Newport News.
Dr. D. C. Smith, University.
Dr. H. C. Spalding, Richmond.
Dr. James B. Stone, Richmond.
Dr. E. L. Stubbs, Newport News.
Dr. E. H. Terrell, Richmond.
Dr. A. E. Tureman, Richmond.
Dr. A. W. VanLandingham, Langley Field.
Dr. Samuel A. Vest, University.
Dr. Thos. M. Vorbrinck, Norfolk
Dr. W. W. Waddell, Jr., University.
Dr. R. H. Walker, Martinsville.
Dr. K. K. Wallace, Norfolk.
Dr. T. J. Williams, University.

Married.

Dr. George Hatcher Snead, Richmond, and Miss Anne Hurtt Ross, Onley, June 28th. Dr. Snead is an alumnus of the Medical College of Virginia.

Major John Tabb Walke, MC., U.S.A., and Miss Evelyn Chamblin Murrell, both of Richmond, July 6. Major Walke is a graduate of the Medical College of Virginia, class of 1940. He has recently returned from overseas duty in the Indo-china area.

Dr. Ashby Turner Richards, Harrisonburg, and Miss Evelyn Rosaline Suter, Staunton, June 14. Dr. Richards is a graduate of the Medical College of Virginia, class of December, 1943, and is now serving his internship at the U. S. Marine Hospital in New Orleans.

Lt. Joe Lee Frank, Jr., MC., AUS., Richmond, and Miss Barbara Olive Bloxam, Roxboro, N. C., July 14th. Lt. Frank is a graduate of the College of Physicians and Surgeons of Columbia University, New York.

McGuire Hospital to Be First Stop Casualty Base.

The new 1800-bed McGuire Hospital in Richmond will act as a receiving hospital for casualties from the European and Mediterranean war theatres. Urgent medical attention will be given and, after a short stay, evacuees will be reassigned to hospitals nearer their homes. Woodrow Wilson Hospital in Staunton will handle neuropsychiatric cases "if it specializes in anything". To make room in the general hospitals, army station hospitals at Ft. George G. Meade, Md., and Camp Lee, Va., have been designated regional hospitals for the ASF. They will perform the same service as general hospitals but for men assigned to the zone of the interior, i.e., posts in this country. The Lee Hospital may also receive any overflow from McGuire.

Dr. Nelson Mercer,

Blacksburg, has been named chief medical officer in the tuberculosis division of the Gallinger Mu-

nicipal Hospital in Washington. He succeeds Dr. Charles P. Cake who resigned early this year. Dr. Mercer is a native of Richmond where he practiced for sometime before going to Chestnut Hill, Pa., where he was resident physician at the Home for Consumptives. For the past year he has been medical officer at Virginia Polytechnic Institute.

Physician-Artists' Prize Contest.

The American Physicians Art Association, with the cooperation of Mead Johnson & Company, is offering an important series of War Bonds as prizes to physicians in the armed services and also physicians in civilian practice for their best artistic works depicting the medical profession's "skill and courage and devotion beyond the call of duty".

Announcement of further details will be made soon by the Association's Secretary, Dr. F. H. Redewill, Flood Building, San Francisco, Cal.

Dr. Huston St. Clair,

Tazewell, has been named chairman of the vocational education study committee of the State School Study Commission. He has also accepted the State chairmanship of the Committee for Economic Development of the CED.

Dr. C. L. Riley,

Winchester, having recently successfully passed the examinations given by the American Board of Obstetrics and Gynecology, has received notice of certification by the Board.

Dr. Riley is now a Lieutenant in the Medical Corps, USNR, and is stationed in Richmond.

Second Annual Summer Clinic.

The Fourth District Medical Association and the Southampton County Medical Society were sponsors of the second annual summer clinic held at the Raiford Hospital, Franklin, on July 26. The program was as follows: The Use of the Different Insulins in Diabetes by Dr. William R. Jordan; Advancements in Pharmacology—Penicillin and New Sulfonamides by Dr. Harvey Haag; and Tropical Diseases: Our New Responsibilities by Dr. Harry H. Henderson. All speakers are of Richmond.

At the Clinic to be held on August 30, there will be a symposium on Pediatrics, and on September 29, the discussion will be on Endocrine Problems.

The American Gynecological Society

Held its annual meeting at Hotel Hershey, Hershey, Pa., the latter part of June, a very interesting program being presented. The presidential address by Dr. George W. Kosmak of New York City was entitled "The Place of Woman in the Changing World". In the business session, Dr. Edward A. Schumann of Philadelphia was elected president; Drs. Norris W. Vaux of Philadelphia and James C. Masson of Rochester, Minn., vice-presidents; and Dr. Howard C. Taylor, Jr., of New York and Dr. Philip F. Williams of Philadelphia, secretary and treasurer, respectively. Time and place of the next meeting to be announced later.

International College of Surgeons.

The program of the Ninth Annual Assembly of the United States Chapter of the College will be devoted to War, Rehabilitation and Civilian Surgery. The sessions will be held at the Benjamin Franklin Hotel in Philadelphia, October 3-5. The convention opens on the morning of the 3rd and in the evening "Service Night" will be held at which time the guest speakers will be prominent members of the Army, Navy and Veterans Administration. More than fifty prominent surgeons and others engaged in the work of rehabilitation and occupational therapy will present twenty-minute papers during the morning and afternoon sessions. There will also be a tour of hospitals and attendance at clinics. Outstanding scientific exhibits are planned and more than 250 feet of panel space has been set aside for them.

The medical profession is invited to attend the Assembly and its sessions. Information with regard to hotels, etc., may be obtained from Dr. Benjamin Shuster, Philadelphia.

Dr. R. Finley Gayle,

Richmond, was elected to membership on the National Committee for Mental Hygiene at the meeting of the Board of this organization in New York City, early in June.

American College of Chest Physicians.

At the annual meeting held in Chicago, June 10-12, Dr. Dean B. Cole, Richmond, was re-elected as a Governor of the College for a term of three years. Others who attended from Virginia were: Drs. R. H. Walker, Martinsville; R. B. Grinnan, Jr., Norfolk; and E. C. Harper, Richmond.

Promotions in the Service.

The following promotions for our members in the Service have recently been noted:

Dr. Harry J. Warthen, Jr., Richmond, to Lieutenant Colonel, U. S. Army.

Dr. H. B. Yeatts, Danville, to Lieutenant Commander, U. S. Naval Reserve.

Dr. D. Coleman Booker, Richmond, to Captain, U. S. Army.

Dr. C. B. Bowyer,

President of the Medical Society of Virginia, who was recently confined to Johnston Memorial Hospital, Abingdon, on account of illness, has improved sufficiently to return to his home at Stonega, and is resuming his work by degrees.

Dr. William L. Davis,

Recently of Norfolk, is now engaged in special work at the Margaret Hague Maternity Hospital in Jersey City, N. J.

Medico-Legal Conference and Seminar.

I. Conference

The Massachusetts Medico-Legal Society in conjunction with the medico-legal departments of Harvard, Boston University and Tufts Medical Schools has arranged for an all-day conference to be held at the Mallory Institute of Pathology, Boston City Hospital, on Wednesday, October 4, 1944. It will include lectures, demonstrations, and informal discussions concerning many subjects in legal medicine, particularly stressing some of the more recent procedures. This meeting will be open to any registered physician, lawyer, police official, senior medical student or other medical investigator who may be interested and care to register. No limit in number has been made. There will be no fee for registration. While advance application is not essential, it would be helpful to those arranging the conference if notice of intention to attend be sent prior to October 1 to Dr. W. H. Watters, Department of Legal Medicine, Harvard Medical School, Boston.

II. Seminar

The Harvard Medical School, Courses for Graduates, with the co-operation of the Medical Schools of Boston University and Tufts College offers a seminar in Legal Medicine to occupy the entire week of October 2-7, inclusive. It is planned particularly for medical examiners and coroners physicians but will be open to any other suitable graduate of an approved medical school.

The course will be practical rather than theoretical and will consist of autopsy demonstrations, technique and interpretation of laboratory tests, study of the day-by-day cases of a medical examiner, round table conferences, and the many subjects now included in the widening field of legal medicine. In order that each participant may receive the maximum benefit, the enrollment has been limited to fifteen. For the seminar the fee is $25. Application should be made on or before October 1 to Harvard Medical School, Courses for Graduates, 25 Shattuck Street, Boston 15, Massachusetts.

Wanted.

Registered technician for office work. Hours 9-5, Thursday and Saturday afternoons off. No night nor Sunday work. Salary $200.00. Reply, with references, to "LFH", care this journal, 1200 East Clay Street, Richmond 19. (*Adv.*)

Obituaries

Dr. William A. Harris,

Prominent physician of Spotsylvania, died May 25. He was sixty-six years of age and a graduate of the Medical College of Virginia in 1901. Dr. Harris served for three terms in the General Assembly as a delegate from Fredericksburg and Spotsylvania County. He was formerly county coroner, secretary of the Board of Public Roads, and County Health Officer, as well as a member of the Board of Visitors of VPI. Dr. Harris was a member of the Medical Society of Virginia. His wife and several children survive him.

Dr. William Alexander Lambeth,

A member of the faculty of the University of Virginia for more than forty-five years and a leading figure in Southern and intercollegiate athletics, died June 24. He received his medical degree from the University of Virginia in 1892 and served as professor of materia medica and hygiene and as head of the department of physical education and director of athletics. Dr. Lambeth was widely known for the thirty years he guided Virginia athletic teams to national prominence. He had served in national and international athletic organizations.

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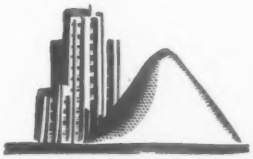
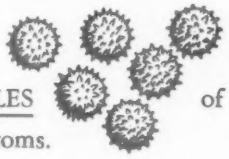
Hay Fever facts and figures

The annual crop of ragweed pollen in North America weighs more than 2 BILLION POUNDS.



A single teaspoon holds more than 1 BILLION PARTICLES of ragweed pollen.



As few as 6 PARTICLES of ragweed pollen can produce hay fever symptoms.

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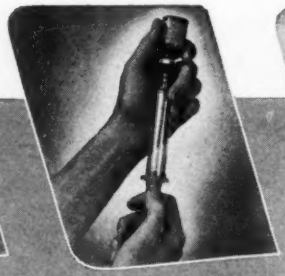
Penicillin-C.S.C.—available as penicillin calcium as well as penicillin sodium—is packaged only in rubber-stoppered serum-type vials containing 100,000 Oxford Units. The vials are used in preference to sealed ampuls because they make for greater convenience in storing the solution and because they lessen the danger of contamination after the solution is made.

Only vials of 100,000 units are offered at present because experience designates them as the most advantageous size. If there IS a factor in therapy which may undermine or lessen the remarkable therapeutic efficacy of penicillin, it may be underdosage. Even if therapy is instituted late in the course of the disease, penicillin in many instances will prove effective if adequately high dosage is used for the proper length of time.

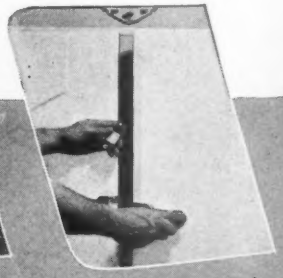
In the conditions so far explored and reported, effective dosage in some instances will be less than 100,000 units per day; in many instances it may have to be several times this amount Hence in a large percentage of cases the Penicillin-C S.C. vial of 100,000 units will prove most advantageous.

The convenience of the vial will be readily appreciated. After removal of the tear-off portion of the aluminum seal, sterilize the exposed surface of the rubber stopper

For the usual concentration (5000 Oxford Units per cc.) inject 20 cc. of physiologic salt solution into the vial in the usual aseptic procedure.



Invert the vial and syringe (with needle in vial), and withdraw the amount of penicillin solution required for the first injection.



Store vial with remainder of solution in refrigerator. Solution is ready for subsequent injections during the next 24 hours.

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IS SUPPLIED ONLY IN 100,000 OXFORD UNIT VIALS

in the customary manner, inject into the vial 20 cc. of pyrogen-free, sterile physiologic salt solution; without removing the needle invert vial and withdraw as many cc. of this 5000 units per cc. solution for the injection that is to be made immediately; store the vial with its remaining solution in the refrigerator—it is ready for use when the next injection is to be made.

The concentration withdrawn from the vial is 5000 units per cc. If a lower concentration is desired, modification is easily accomplished.

If you have not as yet received a copy of the "Penicillin-C.S.C. Therapeutic Reference Table," showing dosages, modes of administration, and duration of treatment required in the various infections in which penicillin is indicated, write for a complimentary copy now. You will find it a valuable aid in familiarizing yourself with penicillin therapy.

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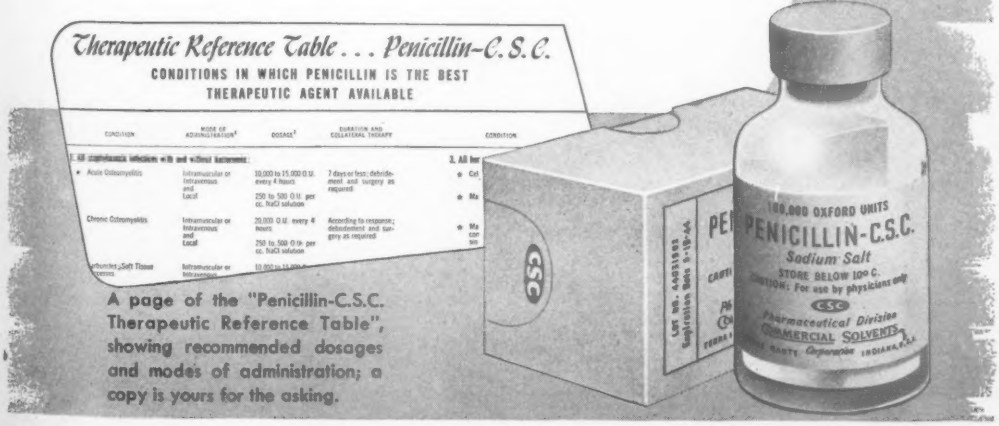
Therapeutic Reference Table . . . Penicillin-C.S.C.
CONDITIONS IN WHICH PENICILLIN IS THE BEST THERAPEUTIC AGENT AVAILABLE
CONDITION
MODE OF ADMINISTRATION
DOSAGE
DURATION AND COLLATERAL THERAPY
CONDITION
Acute Osteomyelitis
Intramuscular or intravenous and Local
10,000 to 15,000 O.U. every 4 hours
250 to 500 O.U. per cc. NaCl solution
7 days or less; debridement and surgery as required
Chronic Osteomyelitis
Intramuscular or intravenous and Local
20,000 O.U. every 4 hours
250 to 500 O.U. per cc. NaCl solution
According to response; debridement and surgery as required
100,000 OXFORD UNITS
PENICILLIN-C.S.C.
Sodium Salt
STORE BELOW 10° C.
CAUTION: For use by physicians only
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TERRE HAUTE, INDIANA, U.S.A.

A page of the "Penicillin-C.S.C. Therapeutic Reference Table", showing recommended dosages and modes of administration; a copy is yours for the asking.

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Component and Other Medical Societies in Virginia

(Officers and Others are Requested to Notify the **Monthly** of Changes)

SOCIETY	PRESIDENT	SECRETARY	TIME OF MEETING
Accomac County	O. R. Fletcher, Sanford	**J. C. Doughty, Onancock**	Quarterly.
Albemarle County	W. W. Waddell, Jr., University	W. R. Mason, University	
		Charlottesville	Monthly.
Alexandria City	S. H. Williams, Alexandria	**Alfred Abramson, Alexandria**	Monthly.
Alleghany-Bath Counties	L. A. **Houff, Clifton Forge**	R. P. **Hawkins, Clifton Forge**	Bi-Monthly.
Amelia County	J. M. Habel, Jetersville	**J. L. Hamner Mannboro**	
Arlington County	J. E. Payne, Arlington	J. A. Cope, Arlington	January, April, July, August.
Augusta County	A. M. McLaughlin, Waynesboro	**George Kinser, Waynesboro**	February, May, August, November.
Bedford County	M. P. Rucker, Bedford	W. V. Rucker, Bedford	Quarterly.
Botetourt County	L. A. **Micou, Eagle Rock**	E. B. Morgan, Fincastle	
Charlotte County	C. M. Nicholson, Charlotte C. H.	J. R. Bailey, Keysville	
Clinch Valley Med. Society	C. H. Henderson, Norton	C. B. Bowyer, Stonega	Spring and Fall.
Culpeper County	J. L. Stringfellow, Culpeper	O. K. **Burnette, Culpeper**	
Danville-Pittsylvania	J. J. Neal, Danville	P. W. Miles, Danville	
County Division	H. H. Hamner, Chatham	G. V. Thompson, Chatham	
Dickenson-Buchanan Counties	J. C. Trivett, Page	T. C. Sutherland, Haysi	
Elizabeth City County	R. H. Wright, Phoebus	J. Lee Mann, Hampton	
Fairfax County	E. S. Waring, Fairfax	**Alice Keisaling, Falls Church**	February, May, August, November.
Fauquier County	Stewart McBryde, Manassas	J. Frank Folk, Warrenton	Monthly.
Floyd County	S. T. Yeatts, Floyd	F. C. Bedsaul, Floyd	
Fourth District and Southside	T. S. Jennings, Waverly	C. E. Martin, Emporia	5 times a year.
Fredericksburg Med. Assn.	F. C. Pratt, Fredericksburg	L. A. Busch, Fredericksburg	Monthly.
Halifax County	L. P. Bailey, Nathalie	W. C. Brann, South Boston	
Hanover County	J. A. Wright, Doswell	J. D. Hamner, Ashland	Bi-Monthly.
Hopewell Med. Assn.	A. T. Brickhouse, Hopewell	W. M. Phipps, Hopewell	
Isle of Wight County	Rea Parker, Smithfield		
James River Med. Society	T. L. Driscoll, Columbia	**Garland Dyches, Dillwyn**	Quarterly.
Lee County	J. H. Dellinger, Pennington Gap	C. H. Henderson, Bonny Blue	
Loudoun County	W. O. Bailey, Leesburg	G. H. Musgrave, Leesburg	Monthly.
Louisa County	H. G. Byrd, Louisa	E. B. Pendleton, Cuckoo	
Lynchburg Academy	S. H. Rosenthal, Lynchburg	L. R. O'Brian, Jr. Lynchburg	Monthly.
Mecklenburg County	H. H. Braxton, Chase City	W. W. Wilkinson, LaCrosse	
Med. Assn. Valley of Virginia	T. Rolston, New Hope	**H. W. Miller, Woodstock**	September.
Med. Soc. Northern Virginia	C. O. Dearmont, White Post		
Medical Society of Virginia	C. B. Bowyer, Stonega	**Agnes V. Edwards, Richmond**	Richmond, Oct. 23-25, 1944.
Mid-Tidewater Med. Society	H. A. Tabb, Gloucester	M. H. Harris, West Point	January, April, July, October.
Nansemond County	E. C. Joyner, Suffolk	H. D. Crow, Suffolk	
Nelson County	Lyddane Miller, Amherst	J. F. Thaxton, Tye River	
Norfolk County	Claiborne Willcox, Norfolk	L. B. Scott, Norfolk	Semi-Monthly
Northampton County	J. M. Lynch, Cape Charles	W. C. Henderson, Nassawadox	Quarterly.
Northern Neck	M. C. Oldham, Kilmarnock	Lee S. Liggan, Irvington	Spring and Fall.
Orange County			
Patrick-Henry Counties	R. H. Walker, Martinsville	T. H. Dickerson, Martinsville	January, April, July, October.
Piedmont Med. Society	Percy Harris, Scottsville	E. D. Davis, Crozet	
Princess Anne County	R. W. Woodhouse, Va. Beach	R. A. Stata, Oceana	
Richmond Academy	E. H. Terrell, Richmond	Mary N. Godbold, Richmond	2nd and 4th Tuesdays.
Roanoke Academy	L. D. Keyser, Roanoke	D. S. Garner, Roanoke	1st and 3rd Mondays.
Rockbridge County		E. P. Tompkins, Lexington	
Rockingham County	H. Grant Preston, Harrisonburg	Chas. C. Powel, Harrisonburg	2nd Mondays, Feb., May, Aug., Nov.
Russell County	T. G. Smith, Lebanon, R. D.	W. C. Elliott, Lebanon, R. D.	
Scott County	C. R. Fugate, Clinchport		
Seaboard Med. Assn.	M. A. Pittman, Wilson, N. C.	**C. P. Jones, Newport News**	Wilson, N. C., 1944.
Southampton County	Morgan Raiford, Franklin	W. T. McLemore, Courtland	April and September.
South Piedmont Med. Society	Bernard H. Kyle, Lynchburg	F. O. Plunkett, Lynchburg	Spring and Fall.
Southwestern Virginia	A. B. Graybeal, Marion	G. C. Williams, Pearisburg	April and September.
Tazewell County	J. W. Witten, N. Tazewell	J. A. Robinson, Richlands	
Tri-State Med. Assn. Va. & N. C.	K. B. Pace, Greenville, N. C.	J. M. Northington, Charlotte, N. C.	Columbia, S. C. 1945
Va. Orthopedic Society	J. B. Dalton, Richmond	J. T. Tucker, Richmond	Richmond, Oct. 23-25, 1944.
Va. Pediatric Society	E. A. Harper, Lynchburg	Emily Gardner, Richmond	Richmond, Oct. 23-25, 1944.
Va. Peninsula Acad. Med.	W. R. Payne, Newport News	R. H. Wright, Phoebus	3rd Mondays.
Va. Radiological Society	C. W. Eley, Norfolk	E. L. Flanagan, Richmond	Richmond, Oct. 23-25, 1944.
Va. Soc. Chest Physicians	F. B. Stafford, Sanatorium	E. C. Harper, Richmond	
Va. Society of Obst. & Gyn.	A. L. Carson, Richmond	L. L. Shamburger, Richmond	Richmond, Oct. 23-25, 1944.
Va. Society of O. L. & O.	James R. Gorman, Lynchburg	Thos. E. Hughes, Richmond	
Va. Urological Society	T. B. Washington, Richmond	W. W. Koontz, Lynchburg	Richmond, Oct. 23-25, 1944.
Warwick County	Paul Hogg, Newport News	E. D. Blechman, Newport News	Monthly.
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Wise County	Glen Foster, Stonega	W. B. Barton, Stonega	Bi-Monthly.

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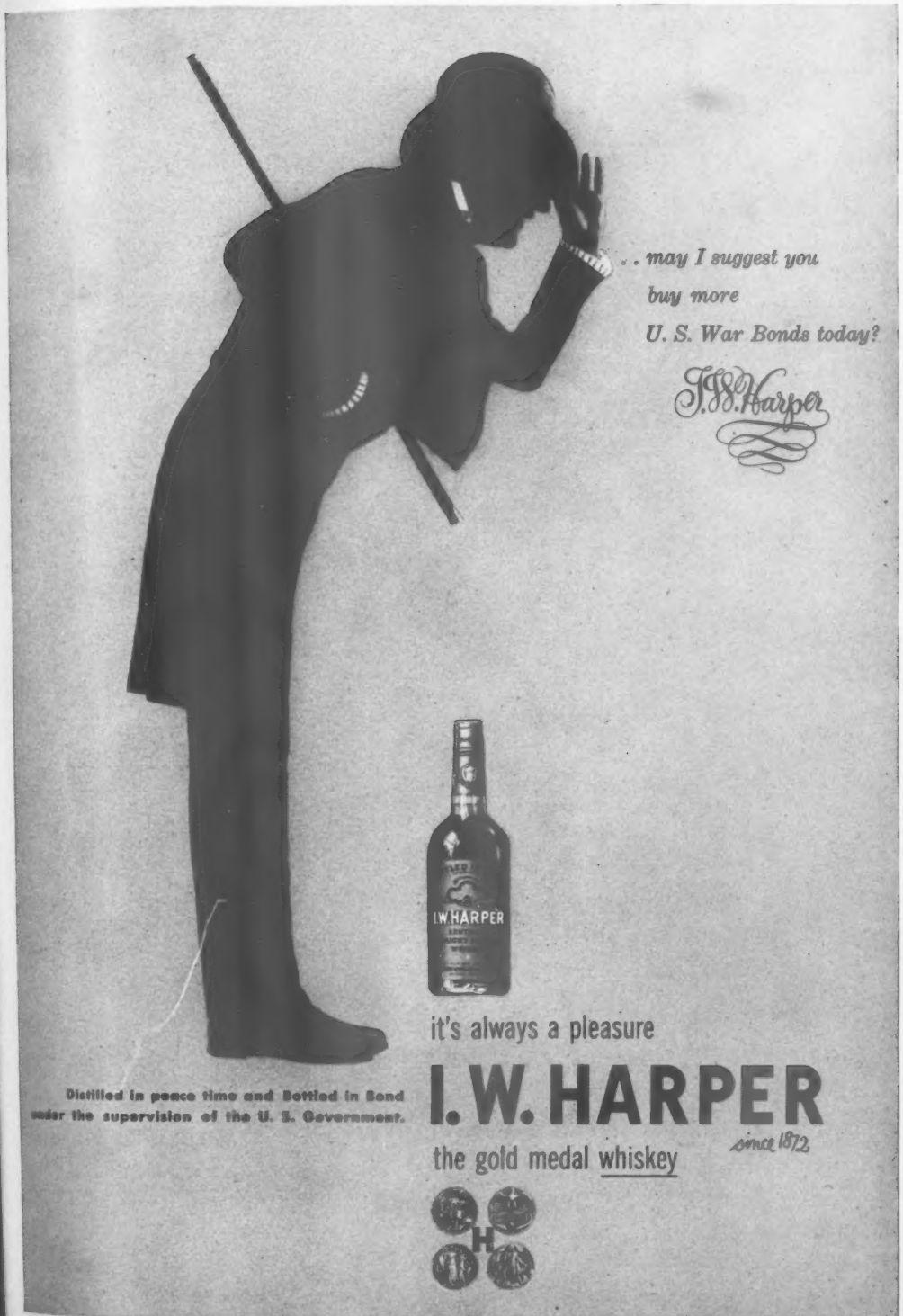

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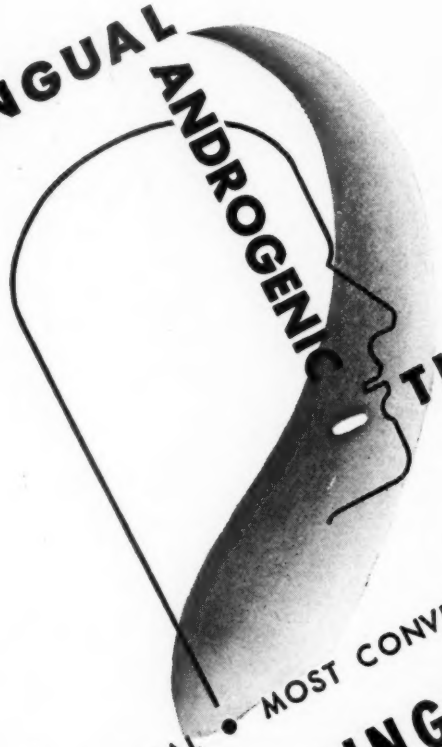

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Small, hard pressed wafers, especially designed for systemic absorption via the oral mucous membranes, thus side-tracking the liver where partial inactivation of ingested methyltestosterone is known to take place.[1]

Effective in smaller doses, e.g. 1/2 to 2/3 the amount required when methyltestosterone is swallowed.[2]

Cost of treatment with METANDREN LINGUETS is considerably less than when androgens are administered by injection, implantation or ingestion.

[1]Biskind, G. R.: Proc. Soc. Exper. Biol. & Med. 43:259, 1940. Burrill, M. W. and Greene, R. R.: Endo. 31:73, 1942.

[2]Lisser, H. and Curtis, L. E.: J. Clin. Endo. 3:389, 1943. *Trade Mark Reg. U. S. Pat. Off.

a Ciba Product

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Facts for the patient inquiring about the

SAFETY

OF INTERNAL MENSTRUAL PROTECTION

Tampax menstrual tampons are more than merely *adequate* for catamenial protection...they possess a wide margin of safety, particularly on prolonged use.

Careful and extended research by authorities in different parts of the country—involving studies on bacterial flora, hydrogen ion concentration, vaginal mucosal biopsies, glycogen determinations and gross examinations in hundreds of cases—has failed to reveal any untoward results from the regular use of this form of menstrual hygiene.

For instance, one investigator[1] reports, "By exact research in 218 women who wore tampons regularly during their menstruation for one year and over, no production of irritation or discharge, vaginitis or cervicitis was found."

Another[2] states that, in 110 subjects using tampons throughout each period for a minimum of one year to a maximum of two years, "there was no evidence of any irritation of the cervix or vagina by the tampon."

A third clinician[3] (with a series of 21 subjects) writes that "no evidence was observed of any infection carried by the tampons."

Finally, the general consensus would seem to indicate that intravaginal menstrual protection will not cause blocking of the flow or cramps—rather that "tampons actually acted as a wick to draw away the blood from the cervix."[1]

Thus, Tampax can be soundly recommended to patients of menstruating age—on the basis that "the evidence is conclusive that the tampon method of menstrual hygiene is safe, comfortable and not prejudicial to health."[4]

(1) West. J. Surg., Obst. & Gyn., 51:150, 1943. (2) Am. J. Obst. & Gyn., 46:259, 1943. (3) Clin. Med. & Surg., 46:327, 1939. (4) Med. Rec., 155:316, 1942.

TAMPAX *accepted for advertising by the Journal of the American Medical Association*

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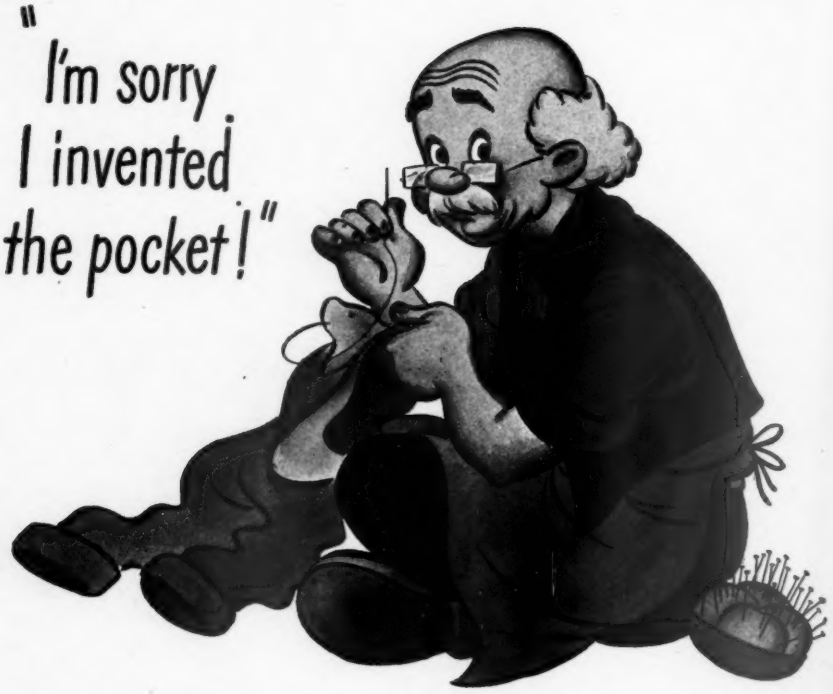

IF I HAD KNOWN that some Americans would be using pockets to hold all the extra money they're making these days, I never would have invented them.

Pockets are good places to keep hands warm.

Pockets are good places to hold keys . . . and loose change for carfare and newspapers.

But pockets are no place for *any* kind of money except actual expense money these days.

The place—the *only* place—for money above living expenses is in War Bonds.

Bonds buy bullets for soldiers.

Bonds buy security for your old age.

Bonds buy education for your kids.

Bonds buy things you'll need later—that you can't buy now.

Bonds buy peace of mind—knowing that your money is in the fight.

Reach into the pocket I invented. Take out all that extra cash. Invest it in interest-bearing War Bonds.

You'll make me very happy if you do.

You'll be happy too.

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They stand alone in sadness and disappointment...women whose hopes have been blighted by repeated spontaneous abortions due to corpus luteum hormone deficiency.

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How to use DRYCO—New Improved DRYCO is quickly soluble in cold or warm water.

Prescribe one leveled tablespoonful per pound of body weight daily, plus sufficient carbohydrate to meet caloric needs. (One tablespoon DRYCO supplies 31½ calories.)

DRYCO is made from spray-dried, superior quality whole milk and skim milk. It supplies 2500 U.S.P. units of vitamin A and 400 U.S.P. units of vitamin D per reconstituted quart. For information, write Borden's Prescription Products Division, 350 Madison Ave., New York 17, New York.

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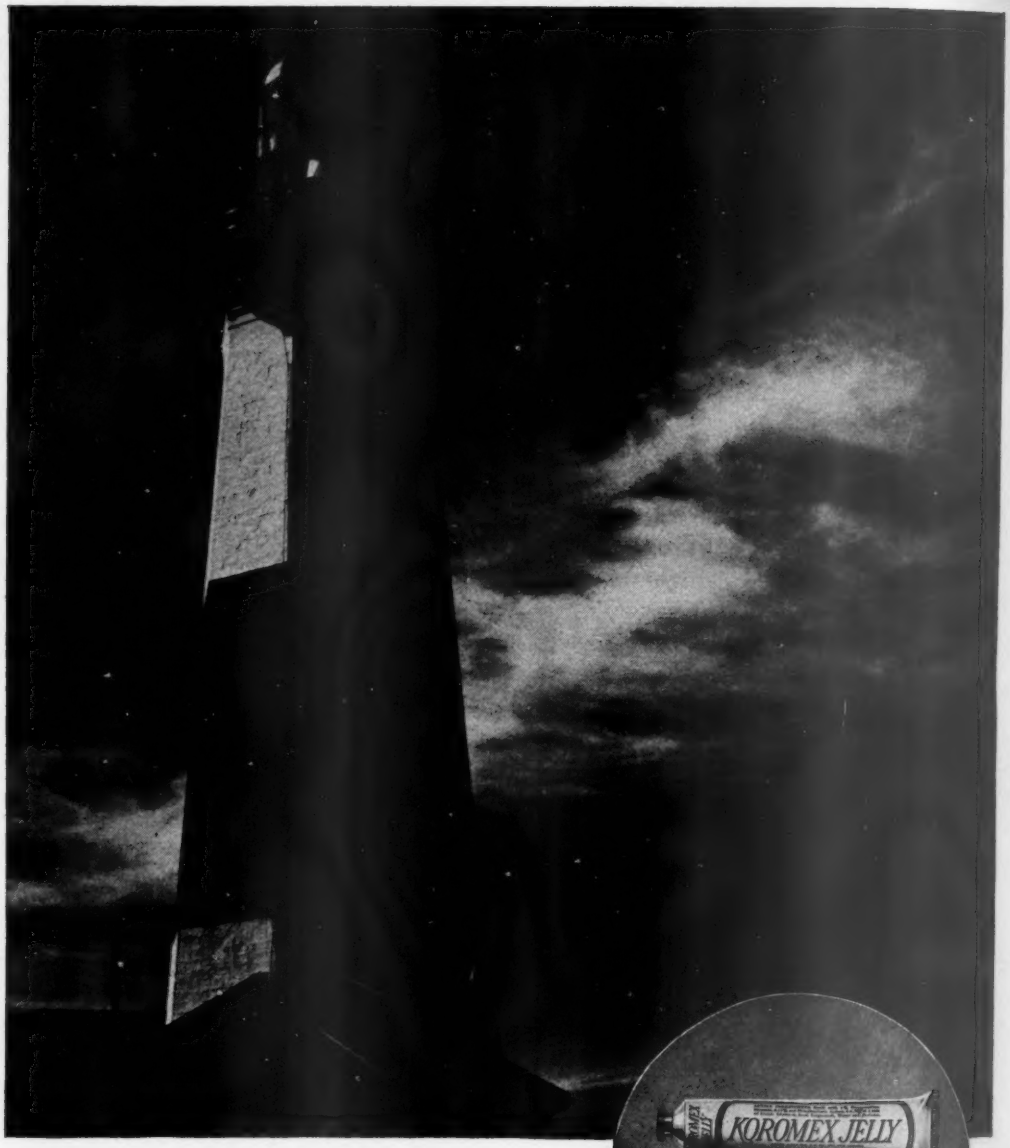

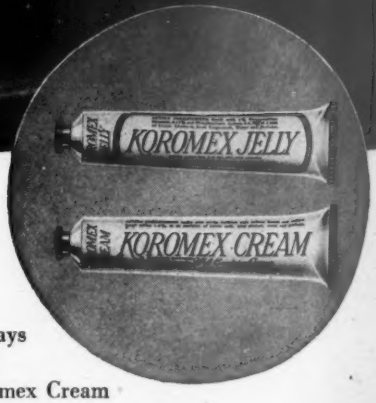

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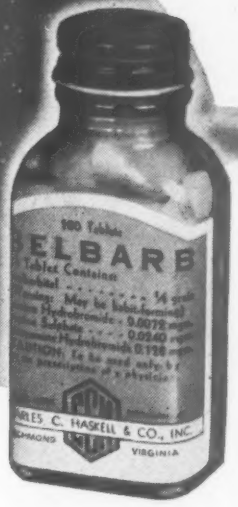

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For the reduction of edema, to diminish dyspnoea and to strengthen heart action, prescribe Theocalcin, beginning with 2 or 3 tablets t. i. d., with meals. After relief is obtained, the comfort of the patient may be continued with smaller doses. Well tolerated.

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Calibration 300 mm.

Size 38½" high x 11½" wide at base. EXACTILT Scale permits maximum reading efficiency from standing or sitting position.

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Die-cast Dowmetal (Magnesium). Base die-cast in zinc.

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The promise of penicillin . . . precious, life-saving antibiotic derived from *Penicillium notatum* . . . will not be fully realized until this drug is available in sufficient quantities to work its miracles in every city, town, and hamlet in the country.

Cheplin Biological Laboratories are actively engaged in the production of penicillin and are making intensive efforts to increase its output to the point where all restrictions on its civilian use can be removed. We are doing our utmost to speed the day when this drug will be found in every physician's bag and every pharmacist's prescription room.

"ACCEPTED STANDARDS AT AN ACCEPTABLE COST"

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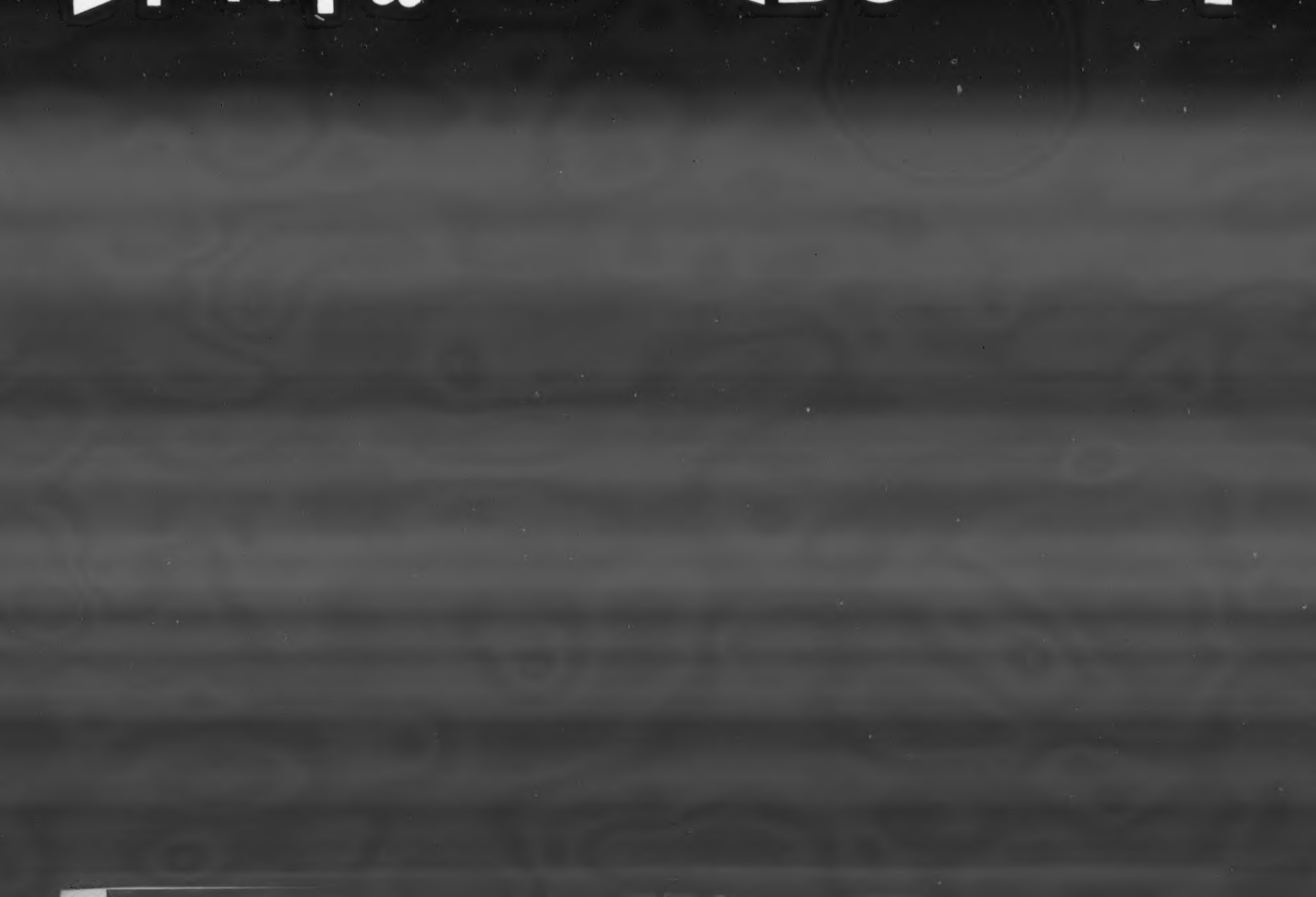
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